DECEMBER, 1951

The Review of Gastroenterology

OFFICIAL PUBLICATION

NATIONAL GASTROENTEROLOGICAL ASSOCIATION

Results of Treatment of Bleeding Peptic Ulcer

Clinical Appraisal of Liver Function Tests

Giant Echinococcus Cyst of the Spleen

Primary Ulcer of the Jejunum

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Seventeenth Annual Convention

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VOLUME 18 NUMBER 12

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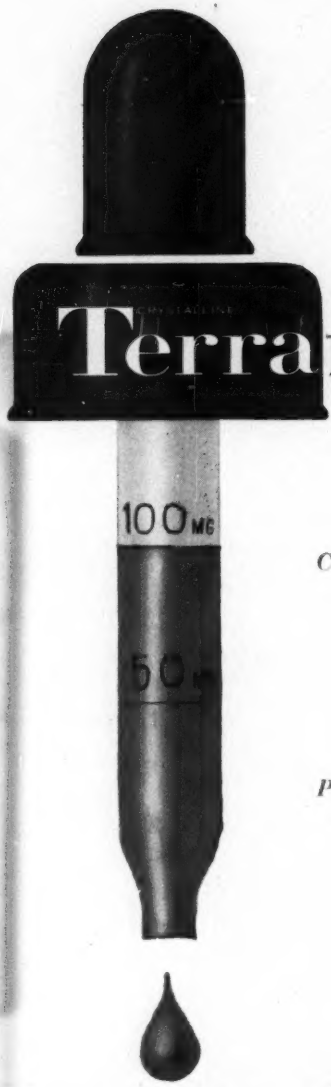

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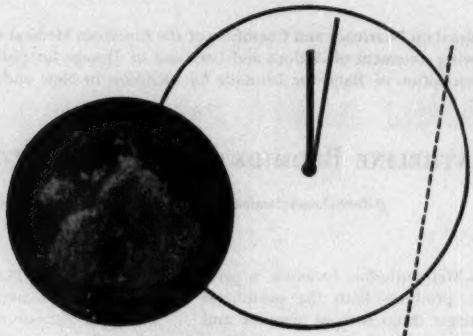

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(INCORPORATING THE AMERICAN JOURNAL OF GASTROENTEROLOGY)

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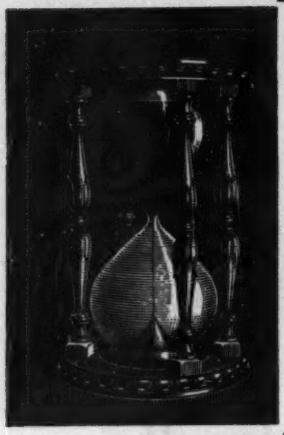

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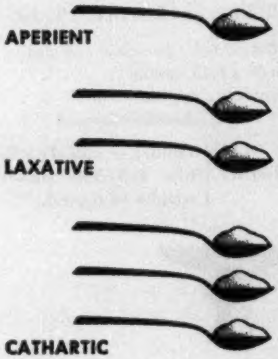

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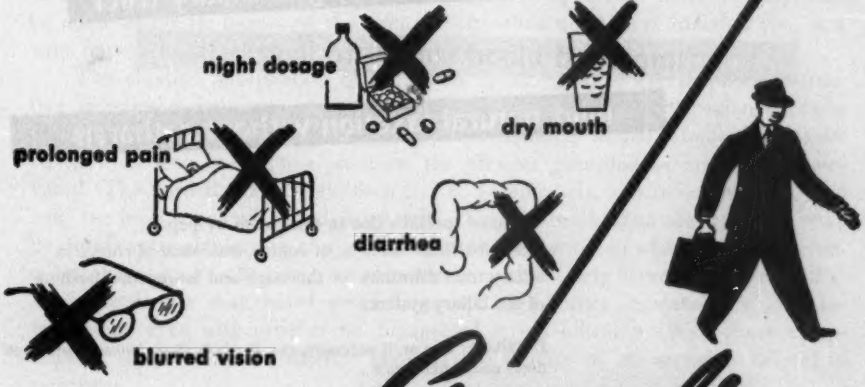

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1. Brauhaus, H. C., Akre, O. H., and Eyerly, J. B.: *Gastroenterology,* 16:172, Sept., 1950.

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The Review of Gastroenterology

(Incorporating the American Journal of Gastroenterology)

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Volume 18 DECEMBER, 1951 Number 12

CLINICAL APPRAISAL OF LIVER FUNCTION TESTS*†

HENRY A. RAFSKY, M.D., F.A.C.P.
MICHAEL WEINGARTEN, M.D.
and
CHARLES I. KRIEGER, M.D.
New York, N. Y.

It is a well-known fact that the liver is an organ which has many functions. Up to the present time, approximately twenty-five tests have been described which are indicative, each in its limited way, of a disturbance of hepatic function. These tests roughly estimate three processes: 1) the excretory function, 2) the synthesizing function, and 3) injury to the liver parenchyma. The liver function tests are valuable not only in the detection of hepatic impairment, but also as a guide to the effect of therapeutic measures. It is not our intention to enumerate all of the liver function tests, because it is not so important to know how many are available, as it is to evaluate those which are useful in our daily clinical work.

The tests which we propose to discuss are the following: the alkaline phosphatase, cephalin flocculation, thymol turbidity, cholesterol partition, bromsulfalein retention test, urobilin and urobilinogen in the urine, and serum protein determination, and A/G ratio, chemically and by electrophoresis. Mention must also be made of needle biopsy of the liver, which, while not a liver function test, is a very important diagnostic measure, supplementing the liver function tests.

The alkaline phosphatase of the serum is increased in extrahepatic obstructive jaundice due to any cause. A serum level of more than 10 Bodansky units is usually encountered, often increasing proportionately as the jaundice increases. In children with obstructive jaundice, the alkaline phosphatase may not be elevated. The normal range in children is 5 to 14 units. In infectious hepatitis, as a rule, the level is below 10 Bodansky units. The serum phosphatase, however, may be significantly elevated in chronic cholangiolitic hepatitis, in which the obstruction is intrahepatic. Furthermore, an elevated or progressively increasing serum alkaline phosphatase may be of great diagnostic significance in metastatic liver involvement, even with little or no increase of serum bilirubin. With these exceptions, it is true that the alkaline phosphatase activity of the serum is helpful in

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†From the Lenox Hill and Beth Israel Hospitals, New York.
This study was aided by a grant from the U. S. Vitamin Corp. Methischol and Vi-Syneral Therapeutic were supplied through the courtesy of Dr. Louis Friedman.

differentiating jaundice due to extrahepatic obstruction from that due to hepatocellular disease.

The cephalin flocculation and thymol turbidity tests are employed in detecting evidence of parenchymal liver impairment. The cephalin cholesterol flocculation test of Hanger is not necessarily diagnostic of primary liver damage, because other diseases such as virus pneumonia, infectious mononucleosis, congestive heart failure, etc., may produce a postive flocculation test. Positive cephalin flocculation reactions are obtained in practically all cases of acute hepatitis, in the majority of cases of cirrhosis and chronic passive congestion of the liver, and in some cases of hepatic malignancy and in patients with biliary obstruction and ascending infection. The cephalin flocculation test has been found to be a sensitive screening test, and is of value in indicating the presence of incipient or occult damage to liver cells in patients with or without jaundice. It is also of value as a guide in following the progress of acute hepatitis, since the test usually remains positive until the active disease process involving the liver cells has abated.

The cephalin flocculation test does not indicate, quantitatively, the degree of liver impairment or reserve of liver function in such diseases as hepatitis or cirrhosis. The test is of value in a patient with jaundice due to obstruction, as a presumptive indication of secondary damage to the liver cells. The thymol turbidity and the cephalin flocculation tests are positive in more than 90 per cent of cases of infectious hepatitis. The thymol test is usually negative in cases of obstructive jaundice due to extrahepatic obstruction. In infectious hepatitis the thymol turbidity test usually becomes positive at a later date and persists longer than does the cephalin flocculation test. The thymol turbidity test has several technical advantages. It is more easily performed, the solutions are more stable, it can be interpreted in a half-hour, and it is somewhat more specific as a presumptive test for infectious hepatitis. The thymol turbidity is not subject to errors due to aging of the serum or reagent or to sunlight.

The determination of the plasma cholesterol partition is a test which we use routinely. We generally regard 150 to 230 milligrams as normal levels, with cholesterol esters at 65 to 70 per cent and free cholesterol at 30 to 35 per cent. In obstructive lesions of the biliary tract the total cholesterol rises. However, if the obstruction is of long standing and secondary liver damage has occurred, the cholesterol may not be elevated above normal. In prolonged hepatitis there may be marked jaundice and an elevation of the serum cholesterol. In acute hepatocellular damage the cholesterol is usually normal or below normal and the percentage of cholesterol esters may be decreased out of proportion to the lower cholesterol. However, in the cholangiolitic type of hepatitis, the serum cholesterol may be elevated. Hypercholesterolemia is occasionally found in patients with cholelithiasis without obstruction of the common bile duct. The plasma cholesterol concentration and partition are usually normal in uncomplicated portal cirrhosis. Reduced cholesterol levels are the rule in advanced forms of this condition, the decrease occurring chiefly in the ester fraction. A diminished proportion of cholesterol esters in the plasma (normally about 65 to 70 per cent of the total), is

observed in diseases accompanied by hepatocellular damage, with or without a diminution in total cholesterol concentration. This has been observed in acute and subacute hepatic necrosis, phosphorus and chloroform poisoning, arsphenamine hepatitis, and toxic hepatitis, in which the total cholesterol may even be elevated, but the ester fraction is reduced.

Another important liver function test is the bromsulfalein retention test, or as it is commonly referred to, the B.S.P. test. It may be performed by employing either 2 or 5 milligrams of dye per kilogram of body weight. If the 2 milligrams method is used no dye should be retained in the blood after thirty minutes; if the 5 milligrams procedure is employed, a retention of 5 to 8 per cent of the dye in forty-five minutes is regarded as being within normal limits. An increase in the serum bromsulfalein retention takes place if there is parenchymal cell damage of the liver or obstruction to the outflow of bile, as in an obstructive lesion which, by increasing the back pressure in the biliary tract, reduces the excretion of both bile and bromsulfalein, or where there is a decrease in the minute volume of blood flowing through the liver such as occurs in cases of fibrosis of the portal area or cirrhosis or in heart failure so that less dye is presented to the hepatic cells. Normally the injected dye remains almost completely in the blood stream until it is removed in a progressive manner, by the parenchymal cells of the liver and excreted into the bowel and eliminated in the feces. In patients who have no disease of the liver, but who are suffering from shock, hemorrhage, cardiac failure, etc., there may be increased bromsulfalein retention.

The bromsulfalein test is of particular value in the preoperative study of patients with cholelithiasis. It has been found that some degree of retention of dye may occur in obstructive jaundice due to gallstones. It has also been observed that following relief of obstruction, dye retention, although diminishing, frequently persists for a variable period of time after the serum bilirubin concentration has returned to normal. This is probably due to a residual hepatitis which is present in nearly all patients who have suffered from biliary obstruction for any extensive period. Varying degrees of dye retention, up to 100 per cent, may be found in patients with chronic hepatic lesions, as chronic hepatitis, portal cirrhosis, biliary cirrhosis, malaria, hepatic syphilis, carcinoma of the liver and chronic passive congestion of the liver. The majority of observers agree that in portal cirrhosis, particularly, dye retention may occur in the absence of hyperbilirubinemia.

Urobilin and urobilinogen are found in the urine of patients with impaired hepatic function. Absence of urobilinogen from the urine in patients with jaundice is indicative of complete obstruction of the common duct, or of complete suppression of bile pigment excretion by the liver. In calculous obstruction with infection of the bile passages some urobilinogen may appear in the urine. Urobilinuria occurs regularly in the presence of hepatic function impairment, as long as adequate quantities of bilirubin are entering the bowel. Many observers believe that excessive urobilinuria is perhaps the most sensitive single index of the presence of liver dysfunction, except in cases of hemolytic anemia and pernicious

anemia. Excessive urobilinuria occurs commonly in portal cirrhosis even in the early stages of its development. Urobilinuria has been frequently observed in patients with congestive heart failure, and is due to the presence of hepatic damage, functional or organic and usually precedes the development of hyperbilirubinemia. Urobilinuria may also occur in patients with cholecystitis and cholelithiasis in the absence of hyperbilirubinemia as a result of an associated mild hepatitis. This is important in the preoperative study of such patients. Watson has found that 1 to 4 mg. of urobilinogen are eliminated daily in the urine by normal adults. According to the less accurate method employed by Wallace and Diamond, a positive reaction for urobilinogen may be obtained normally with dilutions of urine up to 1 to 20. A positive reaction with dilutions higher than this is considered indicative of the presence of excessive quantities of urobilinogen.

The liver plays an important part in protein metabolism. The serum proteins normally are from 6 to 8 grams per 100 c.c. Hypoproteinemia may be encountered in chronic hepatic diseases such as cirrhosis of the liver, carcinoma of the liver, etc. In patients with diarrhea from sprue, ulcerative colitis, celiac disease, regional ileitis, etc., hypoproteinemia may be observed. The relation of the serum albumin to the globulin, or the A/G ratio is of great clinical significance. The normal ratio is usually considered to be between 1.3 to 1.5. In cirrhosis and hepatitis the albumin is decreased and the globulin fractions are increased resulting in a reversal of the A/G ratio. A moderate to a marked reduction in the total serum protein concentration has been observed in advanced stages of chronic hepatitis and chronic passive congestion, and at times, in acute and subacute hepatitis, acute and subacute hepatic necrosis, and in malnutrition or impaired albumin synthesis. In some instances, particularly in acute forms of liver disease, the serum albumin may be only moderately reduced and the serum globulin increased. This increase in globulin is observed much more commonly in primary hepatocellular disorders than in obstructive jaundice, and in some cases, especially in cirrhosis, may be so great as to more than counterbalance the albumin deficit, the total serum protein concentration being actually increased. In cirrhosis, the increase has been found to occur mainly in the globulin fractions and as determined by electrophoresis, in the gamma globulin fraction. In other types of hepatocellular damage, such as arsenical hepatitis and catarrhal jaundice, the beta and gamma globulin fractions have been found to be increased. The decrease in serum albumin in hepatic disease has been attributed by some, chiefly to coexisting malnutrition, and by others, mainly to interference with regeneration of serum albumin resulting from impairment of liver function.

In discussing the A/G ratio or the relation of the albumin to the globulin, we must emphasize the use of electrophoresis. The A/G ratio as determined by means of electrophoretic studies often does not agree with the A/G ratio as determined chemically by means of salting out methods.

Electrophoresis has in the last eight to ten years assumed great importance in the determination of the distribution of the blood proteins in liver disease. This procedure, devised by Tiselius and perfected by others, provides a means

for a more accurate and more detailed determination of the serum protein fractions. The chemical methods in general use have resulted in values for albumin which are higher than the electrophoretic values. It has been shown that the A/G ratio as measured electrophoretically is approximately 30 per cent lower than that obtained by chemical determination of the same sample. Furthermore, it has been shown by other workers, besides ourselves[1], that there are marked discrepancies between the results of chemical and electrophoretic analysis in various forms of liver dsiease. We have shown that in the acute phase of infectious hepatitis the chemical determinations do not reflect the actual state of the albumin/globulin partition[1]. This has also been shown in patients with peptic ulcer[2]. We found the electrophoretic pattern a great help in patients with acute hepatitis, either of the infectious or homologus serum type. Our electrophoretic experiments were performed with the aid of an apparatus designed for clinical use by one of our co-workers, Dr. Kurt G. Stern.

A discussion of the appraisal of liver function tests is not complete without mention of needle biopsy of the liver. Liver biopsy supplements the liver function tests and is an important part in the diagnostic armamentarium of liver disease. The technic is comparatively simple but certain precautions must be followed. The usual asepsis is necessary; a prothrombin time is done before the liver biopsy and penicillin is administered after it is performed. We prefer to do the needle liver biopsy when the liver is enlarged. Some observers prefer the anterior, others the axillary route. One must be certain that the intestine is not in the pathway of the needle; this may seem elementary, but it can cause real trouble. A Vim-Silverman needle is used. It is advisable to hospitalize the patient for at least twenty-four hours, as infection and bleeding sometimes follow the procedure, no matter how much care is exercised.

As stated above, liver functions tests are helpful not only in the diagnosis of hepatic impairment, but also in the progress of the disease, and in evaluation of treatment. They also act as a guide to preoperative preparation in biliary tract disease.

The following two case reports may be cited to show how liver function tests were helpful in diagnosis. A fifty year old, white female, was admitted to the hospital complaining of intermittent burning pain in the epigastrium for three and one-half months. She subsequently developed nausea, vomiting and anorexia. Her bowels were regular. She lost ten to twelve pounds in two months. Physical examination was not contributory. A gastrointestinal series showed evidence of gastric retention, but no other demonstrable pathology. Her blood count showed a slight anemia, but was otherwise normal. Her sedimentation rate was 39 mm. in one hour. The liver function tests gave the following results: total cholesterol 243; free 79 (32.5 per cent); esters 164; cephalin flocculation 1 plus; thymol turbidity 0.6 units; total protein 6.9; albumin 4.9; globulin 2.0; icterus index 3.7; serum bilirubin 0.35 mg. per cent; *alkaline phosphatase* 18.6; *B.S.P.* 18.5 per cent. On the basis of the increased alkaline phosphatase and B.S.P. a diagnosis of carcinoma of the liver, probably secondary to a carcinoma of the pancreas, was made.

This diagnosis was corroborated at operation. A tumor was found in the region of the head of the pancreas, constricting the first part of the duodenum and adherent to the gallbladder. A posterior gastroenterostomy was done. It is interesting to note that this patient was not jaundiced, notwithstanding the pathological findings. Subsequently she became jaundiced.

The second case is that of a forty-eight year old, white female, who began to have loose bowel movements about one and a half years prior to admission to the hospital on March 20, 1950. There was a weight loss of about 40 pounds. Physical examination was essentially negative. There was no jaundice. Cholecystography showed a normally functioning gallbladder. A gastrointestinal series revealed a deficiency pattern of the small bowel. A barium enema study was negative. The patient weighed 83 pounds. The laboratory examinations on admission were negative, aside from the fact that fat was found in the stools and there was no hydrochloric acid in the undigested gastric contents. It is important to emphasize the fact that her liver function tests at that time were within normal limits. A diagnosis of nontropical sprue was made and the patient was treated on this basis. She was given a high-protein, low-carbohydrate, fat-free diet and intensive vitamin therapy. She gained 40 pounds after being discharged from the hospital, and was well until six weeks prior to her second admission which was on December 11, 1950. She came into the hospital complaining of pain in the right lower chest, which later shifted to the upper right quadrant of the abdomen. Rales were heard over the right lower lobe, but these subsequently disappeared. On physical examination the liver edge was barely palpable and an indefinite resistance was felt in the right upper quadrant of the abdomen. Her sputum was negative for tubercle bacilli. X-ray of the chest was not contributory. A gastrointestinal series did not reveal any pathology and a barium enema was negative. Cholecystography revealed a nonfunctioning gallbladder. The liver function tests at this time were normal except the alkaline phosphatase which on admission was 6.9 and a bromsulfalein retention of 10.5. Several days later the alkaline phosphatase had risen to 10 and the bromsulfalein retention to 19 per cent, and now the cephalin flocculation was 2 to 3 plus. In view of the increasing alkaline phosphatase and B.S.P. a diagnosis of malignancy of the liver, probably secondary to carcinoma of the pancreas, was made. The patient was explored and a carcinoma at the head of the pancreas, with liver metastasis was found. The use of the liver function tests in this manner may be regarded as a "back door approach" to abdominal diagnosis.

In connection with the appraisal of liver function tests, we should like to stress one aspect of the treatment of chronic hepatitis, namely, the use of lipotropic substances. This subject is a controversial one.

Dietary deficiency of such "lipotropic" substances as lipocaic, methionine, choline, inositol and animal protein causes fatty liver and cirrhosis in animals under certain conditions[3, 4]. The evidence relates these events to a defect in phospholipid synthesis by the liver which accumulates fat in large amounts. Phospholipid synthesis is stimulated by administration of choline or of methionine, the latter pro-

viding methyl groups for the synthesis of choline. Some clinicians, however, feel that this fundamental work does not specifically indict deficiency of any of these nutrients in the pathogenesis of liver disease in man, although they admit improper food consumption unquestionably plays an important role. It is generally conceded that diets of persons in whom fatty liver and cirrhosis develop, whether associated with alcoholism or not, are deficient in protein, especially of animal origin. This deficiency included methionine, choline and the other nutrients usually classed in the Vitamin B-complex[5].

Best and Rideout[6] have shown that dogs in which diabetes develops after pancreatectomy, regularly manifest fatty livers and subsequent fibrosis even if maintained on insulin. These hepatic changes may be prevented by the oral administration of whole pancreas, lipocaic (an extract of pancreas), choline, methionine or hydrolyzed protein. Whole protein does not prevent such changes. The pathogenesis of this condition is similar to that which occurs in animals in whom fatty livers develop from direct dietary deficiency of choline and methionine, except that the depancreatized animals become deficient in spite of a diet adequate in whole protein. In the opinion of some clinicians the lipotropic drugs have a definite place in the treatment of chronic liver disease, while others feel that they have no value. We are not going to enter into this controversial issue, but we would like to state our own personal experience. In acute hepatitis the lipotropic substances are not of much value, unless nausea, vomiting and anorexia are persistent, but in chronic liver disease, especially in fatty infiltration, these drugs have been of definite benefit. It is true that they should not be given alone, but as a supplement to vitamin therapy and a highly nutritious diet. We would like to illustrate these facts with the following case reports: Mrs. C. was admitted to the hospital and a diagnosis of cirrhosis of the liver was made. The liver extended six fingers below the right costal margin. The liver function tests corroborated our clinical impression. A liver biopsy on February 6, 1948, showed evidence of fatty liver and cirrhosis. The patient was placed on a high-protein, high-carbohydrate, low-fat diet, intensive vitamin therapy and lipotropic substances in the form of Methischol, which was the product we used in our studies. The patient made an uneventful recovery and a subsequent punch liver biopsy performed on March 25, 1948, showed fibrosis but no fatty infiltration.

Mr. R. complained of severe exhaustion and fatigueability. He drank alcohol very freely for many years. He was overweight and his liver was enlarged. A diagnosis of cirrhosis of the liver was made and the patient was placed on a high-protein, moderate-carbohydrate, low-fat diet with intensive vitamin therapy and Methischol. The patient showed marked clinical improvement. In this connection it is interesting to note that this patient before treatment had a cholesterol of 370 mg. After treatment the cholesterol was 260 mg. While it is true that in cirrhosis the cholesterol as a rule is normal or decreased, during the early stages of cirrhosis or at times during the course of this disease the cholesterol may be increased. What the relationship is between the blood cholesterol and fatty infiltration during the variable course of cirrhosis is a subject which requires further study.

A Mr. S. presented a very similar picture, also complaining of severe exhaustion. Fatigue may often be the chief complaint in early cases of cirrhosis. A diagnosis of cirrhosis of the liver was likewise made in his case. The cholesterol before treatment was 360 mg. This patient was placed on a similar regime and also responded to treatment. His cholesterol after treatment was 240 mg.

Some observers say that improvement in these cases of cirrhosis is due to diet, abstinence from alcohol and vitamin therapy. In the light of our present knowledge our experience is that in chronic hepatitis, with a tendency to fatty infiltration, the lipotropic substances are very useful. A recent editorial in the *Journal of the American Medical Association*[7] goes so far as to suggest that a patient who is a chronic alcoholic and is going out on a week-end bout should fortify himself with lipotropic substances before taking alcohol. It is undoubtedly a clinical fact that many patients with chronic hepatitis have a nutritional deficiency. If they eat well they do not need vitamins or any other supplements. However, experience has shown that these individuals are nutritional problems, irrespective of what history they may give and hence they should be treated accordingly. It is prudent to protect them against their dietetic indiscretion and hence lipotropic substances should be administered daily, even though they are encouraged to eat a high-protein, high-carbohydrate and low-fat diet.

We would like to cite a case report to show how a needle liver biopsy eliminated the necessity for an exploratory operation and revealed the presence of a fatty liver which was successfully treated medically. Mrs. L. G., a fifty-nine year old white female, complained of increasing weakness for three years, which in the past three weeks had become extremely severe. In addition she had an intermittent spiking temperature. She had had occasional swelling of the ankles for many years. There was no alcoholic history. On physical examination, the only positive finding was a liver which was palpable about two fingers below the costal margin. The laboratory findings were: serum bilirubin 0.4 mg; alkaline phosphatase 7.2 units (Bodansky); cephalin flocculation 3 plus; thymol turbidity 3 units; B.S.P. 21.5 per cent retention; the cholesterol was normal. Clinically, the impression was that we were dealing with a possible malignancy. However, it was decided to do a needle liver biopsy before exploring the patient. This was done and the punch biopsy showed evidence of a fatty liver. It was then decided to treat the patient medically. The patient gradually improved and left the hospital feeling fairly well.

There is another aspect of the appraisal of the liver function tests which we would like to emphasize, namely, that pertaining to operations on the biliary tract. Years ago one encountered what was termed the hepatorenal syndrome, even following a comparatively simple cholecystectomy. Technically, from a surgical viewpoint, everything would go very smoothly. Within twenty-four to forty-eight hours after the operation, the patient would suddenly develop oliguria, a high temperature, gradually go into a coma and succumb in a few days. Irrespective of what therapeutic measures were applied, these patients were not saved. This was

a rather puzzling clinical syndrome until we began to evaluate the liver function tests before operation. If abnormal function tests were present, the operation was postponed (unless an emergency existed), until the liver function tests showed marked improvement. Patients were then prepared for biliary tract surgery with the use of parenteral solutions of glucose, saline, vitamins and liver extract. By these procedures, the patients were so prepared that the liver was able to withstand the shock of biliary tract surgery. Fortunately, since these measures have been instituted, we have not seen a case of the so-called hepatorenal syndrome following biliary tract surgery. It is advisable to devote a few days to adequately prepare these patients for surgery.

Summary

1. The evaluation of various liver function tests have been discussed.

2. The importance of liver function tests both from a diagnostic standpoint and therapeutic guide have been outlined.

3. The use of liver biopsy and electrophoresis in conjunction with liver function tests has been stressed.

4. The use of lipotropic substances in the treatment of chronic hepatitis was discussed.

5. Adequate preparation of patients for biliary tract surgery has been emphasized.

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GASTROILEOSTOMY*

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Thirty-two instances of gastroileostomy, a serious surgical error, have been reported in enough detail to permit analysis. This incidence is much lower than actuality since surgeons are not prone to report errors of this magnitude. Of interest is that these cases have been recorded by the surgeons performing the corrective surgery.

The first instance of gastroileostomy mentioned in the literature is that of Judd[4]. The first completely reported case is that of Martin and Carroll[6]. The instances of Mecur[7], Berg[2] reported by Klein, Rivers and Wilbur[11], Kogut and Stein[5], Smith and Rivers[12], Brown, Colvert and Brush[3], Moretz[9], Bailey and Castleton[1], Polivy [10] and Michels, Brown and Crile[8] were the ones collected for review. The cases of Judd and Klein are but mentioned. Two instances of Rivers and Wilbur are not included in this review inasmuch as the patients had gastrojejunostomies with jejunoileostomies although the syndrome produced would be the same as a gastroileostomy. One case of Rivers and Wilbur was reported more completely in a later paper by Smith and Rivers.

Thirty-two instances collected from the literature and the one to be reported are available for review. The time between the performance of the gastroileostomy and of corrective surgery ranged between three months and 25 years. Gastroileostomy without gastric resection was done 26 times while a gastric resection accompanied the error seven times. In the total of 33 patients, a loss of weight occurred in 27 patients, did not occur in 2 and was not stated in 4. Pain in the abdomen was present in 22, hemorrhage in 6, vomiting in 18, which was fecal in character in 6, and diarrhea in 19.

Case Report

C.G. (R-25608), a 53 year old white male was admitted to the Veterans Administration Hospital on June 11, 1950 with persistent diarrhea of about seven months' duration. In November, 1949, a partial gastrectomy was performed elsewhere for a duodenal ulcer causing recurrent epigastric pain and three episodes of hematemesis. The diarrhea was noted almost immediately after operation. The stools were bulky, contained blood and undigested food and numbered eight to thirteen a day. Cramping abdominal pain, relieved by the passage of flatus or stool, was noted occasionally. Edema of the lower legs which extended to the groins started about four months before admission. A considerable loss in weight had occurred.

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The patient was fairly well developed, poorly nourished and drowsy. A severe bronze pigmentation of the skin of the face and arms was present. Numerous rales were heard over the bases of both lungs. The heart sounds were regular and of good quality and murmurs were not heard. The blood pressure was 140/60. Ascites was quite severe. A small hernia was noted in a wound over the right rectus abdominis muscle. A pitting edema extended from the groin to the feet.

The hemoglobin was 69 per cent, the erythrocytes 3,520,000 per cu. mm. and the leucocytes 9,200 per cu. mm. of which 68 per cent were polymorphonuclear neutrophilic leucocytes and 32 per cent lymphocytes. The serum calcium was 8.9 mg. per cent and the inorganic phosphorus was 6 mg. per cent. The total serum

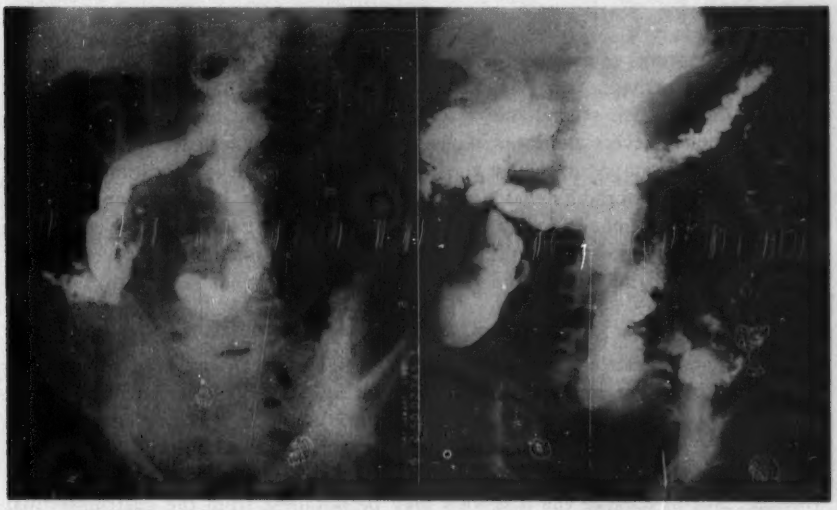

Fig. 1 Fig. 2

Fig. 1—Barium passes directly from the stomach into the terminal ileum through the gastroileostomy.
Fig. 2—Demonstration of gastroileostomy, stomach, terminal ileum and colon.

proteins were 4.2 grams of which 2.2 grams were albumin and 2 grams were globulin with an albumin/globulin ratio of 1.1 to 1. The prothrombin time was 17 seconds with the control 13.5 seconds. The cephalin flocculation test showed a two plus reaction. The roentgenograms of the gastrointestinal tract after the administration of barium showed a gastroileostomy with the anastomosis being about 37.5 cm. from the ileocecal valve (Figs. 1 and 2).

The patient was given a diet rich in proteins and vitamins with the additional administration of liver extract and vitamins. Numerous transfusions of whole blood and blood plasma and infusions containing sodium, potassium, and calcium helped to prepare the patient for operation. Adequate doses of Vitamin K were also given.

Operation was performed on June 30 and an anterior gastroileostomy was found, the anastomosis having been performed about 37.5 cm. from the ileocecal valve. Three thousand c.c. of clear straw-colored fluid were aspirated from the peritoneal cavity. The walls of the gastrointestinal tract were markedly edematous. After the area of the anastomosis was freed, about 5 cm. of the stomach and part of the ileum were removed. A no-loop isoperistaltic posterior gastrojejunostomy was done. The open ends of the ileum were closed and a side-to-side isoperistaltic anastomosis performed. The hernia in the old incision over the right rectus abdominis was repaired. Marked improvement and a gain in weight occurred during the postoperative period. The edema at the ankles was minimal at the time of discharge on August 17. The patient has remained well.

Comment

The symptoms of gastroileostomy are loss of weight, pain of some type in the abdomen, hemorrhage, vomiting, which may be fecal in character, and diarrhea. The syndrome which accompanies gastroileostomy without partial gastrectomy is is usually not as severe and later in onset than when a partial gastrectomy is done. The severity of the syndrome in the former instance will be conditioned by the potency of the pyloroduodenal area. The passage of a large amount of food through the normal channel may permit the patient to have a relatively good state of health with little loss of weight over a long period of time. Pain may be colicky in character or it may be of the severe type similar to that seen with a jejunal ulcer. Hemorrhage usually occurs from an ileac ulcer which was present eight times in the collected series. The ileac ulcers occurred in every instance in patients who had had a gastroileostomy without a partial gastrectomy, a result which is to be expected since removal of the acid-bearing portion of the stomach should help to protect the patient from such ulceration. Vomiting may be very severe in degree and fecal in character. Food passing through a patent pyloroduodenal area may traverse almost the entire small intestine and re-enter the stomach to be vomited. Aid in making the diagnosis may be obtained from the gastrointestinal series since barium may be shown leaving the stomach to enter the terminal ileum. If the pyloroduodenal area is patent, Brown, Colvert and Brush have demonstrated that, in some instances, the stomach will empty itself of barium to refill partially in three to five hours through the misplaced anastomosis. These authors consider this demonstration diagnostically significant.

Gastroileostomy must be differentiated from jejunal ulcer, gastrojejunocolic fistula and vagotomy combined with gastroileostomy. The syndrome resulting from the last of these conditions must be recalled at present, since vagotomy and gastroenterostomy are being used for the treatment of duodenal ulcer in many quarters. The increased frequency of this operation may result in a greater number of gastroileostomies being performed.

The symptoms of jejunal ulcer may appear early or late after operation. Pain may be more severe than that associated with the original gastric or duodenal ulcer. The pain is placed lower in the abdomen and to the left of the um-

bilicus. Hematemesis does not occur as frequently in patients with jejunal ulcer as melena. The gastrointestinal series may show a jejunal ulcer. A satisfactory response to medical treatment is usually not observed in these patients.

The symptoms of jejunal ulcer and ileac ulcer, a complication of gastroileostomy, may parallel each other. The disturbances peculiar to the low anastomosis will aid in the differentiation.

Gastrojejunocolic fistula produces symptoms similar to those encountered in a patient with a gastroileostomy. The interval between operation and onset of symptoms is usually longer in the instances of fistula. If the pyloroduodenal area is patent, a prolonged delay, however, may also be observed in gastroileostomy Loss of weight, good appetite, abdominal pain relieved by passage of stool and diarrhea are usually found in patients with fistula. Considerable aid in the differentiation between gastrojejunocolic fistula and gastroileostomy may be obtained from the barium enema and gastrointestinal series. In fistula, barium may be seen to enter the stomach from the colon. In gastroileostomy, the barium may enter the terminal ileum from the stomach, with occasional re-filling of the stomach in three to five hours, as shown by Brown, Colvert and Brush.

Severe vomiting and diarrhea may be observed in patients who have had a vagotomy and gastroenterostomy performed for the treatment of duodenal ulcer. The history and roentgenograms will help the surgeon make the correct diagnosis.

The error of gastroileostomy can be avoided by careful attention to anatomic detail. Identification of the ligament of Treitz will permit the surgeon to place the anastomosis in the proper place. The left half of the transverse colon must be delivered from the abdomen and traction placed upon the mesocolon. The duodenum should be seen and felt passing posterior to the superior mesenteric vessels to the right side of the abdomen. The inferior mesenteric vein serves as an additional landmark to the left of the ligament of Treitz at the base of the transverse mesocolon. The usual points of anatomic differentiation between jejunum and ileum may be some aid in choosing the site of anastomosis, but if the ligament of Treitz is identified correctly, a malplaced gastroenterostomy will not be performed. Congenital anomalies of the gastrointestinal tract may confuse the surgeon initially but if care is taken to identify the landmarks enumerated, error will not result. If the superior mesenteric blood vessels are posterior to the duodenum, a gross anatomic anomaly is present and the most proximal part of the jejunum must be found by tracing downward from the stomach and duodeum. Various degrees of malrotation of the primary mid-gut loop, particularly where the terminal ileum occupies the left upper quadrant of the abdomen, will also confuse the surgeon.

The patient with a gastroileostomy must be carefully prepared for corrective surgery. A severe deficiency state is usually present and replacement of protein, vitamins and minerals is necessary to achieve a successful result. The operation employed to remove the gastroileostomy will depend first, upon what has previously been done surgically to the patient and second, upon the preference of the surgeon. Disconnection of the stomach from the ileum is the only part of the

operation which must be common to all procedures. In many instances, where the stomach had not been resected, a simple disconnection has resulted in cure. In others, a partial gastrectomy was done. Vagotomy and a correctly placed gastroenterostomy is also a useful corrective procedure. Where gastric resection has accompanied the gastroileostomy, disconnection of the anastomosis, resection of a small portion of the stomach and the involved area of ileum, and the creation of a properly placed anastomosis have served as correction.

Summary

Thirty-three instances of gastroileostomy, including a reported case, have been reviewed. The differential diagnosis is discussed. The anatomical landmarks which will aid in prevention of the performance of a gastroileostomy are presented. The necessary preparation and the methods of operation are discussed.

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HEMORRHOIDS

Etiology, Pathology and Treatment

(Part II)

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Treatment

1. *Prophylaxis and palliation:*—Commonly neglected, prophylactic measures include regulation of diet, alcohol, and bowel habits. Intestinal indigestion, intolerance to fat and carbohydrates, obstipation and constipation, diarrhea with irritating stools, local anorectal allergy, contact dermatitis and histaminosis may all be etiologically significant in hemorrhoidal disease and are amenable to simple corrective measures.

In geriatric practice the prophylactic aspect of anorectal disease is of increasing importance. Active measures directed to proper anal hygiene to prevent local irritation and correction of associated cardiovascular, renal, hepatic genitourinary and gynecological conditions may be recommended.

In infancy periodic dilatation of the constipated or congenitally stenosed anal outlet is a valuable prophylactic measure.

Palliation is primarily directed to the arrest of bleeding and the prevention and early reduction of prolapse. (General corrective measures regarding diet, habits, etc., as advised under prophylaxis should be enjoined.) Constipation with straining at stool may be controlled by diet or simple laxation. Drastic catharsis is to be avoided. Mineral oil is generally considered undesirable for habitual use.

Simple astringent ointments may be useful following defecation. They are preferably introduced digitally by the patient. Despite the popularity of suppositories they are of less palliative value than ointments or other local applications, douches, etc. They may have some placebic effect and may afford a slight degree of dilatation.

2. *Injection treatment of Hemorrhoids:*—The origin of the injection treatment for hemorrhoids is obscure. According to Anderson the method was initally tried in 1869 by Morgan of Dublin who injected a solution of persulfate of iron. This sclerosant had been previously used by Long in 1836 for the treatment of nevi.

The introduction and use of the method in this country in 1871 is accredited to Mitchell of Clinton, Ill., who first used phenol in olive oil. The method, however, had been used by Blackwood as early as 1866. Widely exploited by "itinerant pile" doctors and not withstanding its abuse and complications it was quite popular with the laity but disreputed and disregarded by the general medical profession and surgeons. A controversy arose which provoked several conflicting critical reviews in 1879, notably by Andrews, a professor in the Chicago Medical College who collected some 3,000 cases and concluded that the method, if used cautiously, was as good as any other with the exception of the ligature operation.

In 1882, Kelsey's appraisal of the method as a "surgical procedure of certain definite value" and his emphasis on the fact that the unfavorable results were in large measure due to overly strong solutions of phenol which were not necessary, did much to rescue the injection therapy from quackery.

During the next decade, several contributions "pro and con" concerning the technic and the injection media were made, notably by Shuford in 1887, Doyle and Hoyt in 1900. In 1886, Swinford Edwards in England, published a highly favorable report on the method using 10 per cent phenol.

In 1906, Tuttle, whose opinion was highly esteemed by the profession, concluded that "the method is well worthy of thorough consideration".

In 1913, the introduction by Terrell of quinine and urea as the injection media, together with his favorable reports and his generally accepted conclusions concerning its effectiveness and safety has perhaps more than any other single

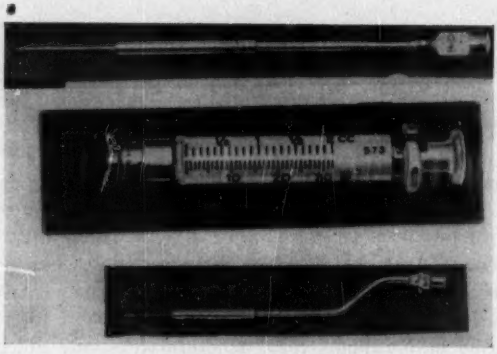


Fig. 12—Types of syringes and needles (Luer-Lok) used for injection.

factor placed the injection treatment on a more rational and scientific basis. Bensuade of Paris also favored quinine and urea in 5 per cent solution.

In England, Edwards, Lockhart-Mummery, Morley, Gabriel, Anderson and others made valuable contributions which have standardized the technic, defined its limitations and emphasized the importance of the proper selection of cases. These authors for the most part popularized 5 per cent phenol in oil as the preferred and safest sclerosant.

Injection of hemorrhoids is now almost universal. It owes its popularity primarily to the fact that the patient may be made asymptomatic even though the hemorrhoids are not cured in the surgical sense. Respite from bleeding and prolapse, a natural aversion to operation and hospitalization, and the anticipation of a painful convalescence, often the case, still weigh heavily in favor of the injection method. So-called ambulatory proctology is an offshoot from the injection treatment.

Historical facts concerning the method have been collected by Anderson in England and Kleiner in this country.

2a. *Selection of cases for injection:*—There is still considerable difference of opinion between the advocates of surgery and the injection treatment, and there are extreme ideas as to what constitutes specific indications for one form of therapy as against the other, or what on the other hand are the contraindications to either method. That the indications are generally not too well defined is reflected by the proctologist who on the one hand treats 85 per cent of his hemorrhoidal patients by injection in contrast to his protagonist who operates the same percentage. A lack of interest in one method is not a legitimate excuse for overenthusiasm in the other.

There are obviously many "extramedical" factors which enter into the selected treatment and the patient often makes the decision by the mere choice of his physician.

At the outset, the proper psychological handling of the rectal patient is sometimes of importance, particularly in those who have heard about the patient "cured" by injection, but who may have complicating anorectal pathology or who are unsuitable for the injection treatment. To the laity all rectal pathology is usually hemorrhoids and the majority, at least in private practice, are already convinced that their treatment and cure is merely to consist of some form of painless injection. It is frequently a delicate and difficult matter to disillusion some patients even at the expense of losing them. The best plan, however, is to place all the facts before such patients before coming to a decision.

The treatment advised should be based and evaluated in terms of the hemorrhoidal pathology, following a complete proctologic or other indicated examination. On this basis internal hemorrhoids may be conveniently divided into those of first, second or third degree.

Clinically considered, first degree hemorrhoids are uncomplicated, do not prolapse, are soft, with no fibrosis and little or no sphincter spasm. The left lateral pile is usually prominent in these cases and bleeding on defecation, is commonly the only symptom of note.

Second degree hemorrhoids are the equivocal group. They are characterized by some degree of prolapse, usually slight, which reduces spontaneously after evacuation. Attenuation of the supporting muscularis mucosa and the extensions of the conjoined longitudinal muscle with greater dilatation of the veins and some degree of interstitial fibrosis following endophlebitic processes and infection, are usually present. Involvement of the external component is sometimes already established and is evidence of chronicity. If the hemorrhoids are complicated or require manual replacement they are usually unsuitable for injection.

Third degree hemorrhoids have lost their supportive matrix and are usually extruded with little straining (Fig. 6). They prolapse completely during defecation. Usually associated with some degree of rectal or so-called anal prolapse, they may assume a polypoid appearance. Their overlying mucosa due to repeated trauma, irritation, congestion, and infection, often presents a granular bleeding surface. Overgrowth with squamous epithelium is common "squamatization" and is an earmark of chronicity. Repeated minute thrombotic and endo-

phlebitic processes result in interstitial fibrotic changes which may be evident and palpable. Secondary enlargement of the corresponding portion of the external hemorrhoidal plexus usually supervenes, forming the mixed or extero-internal hemorrhoid. Skin tags may be present. This local pathology is commonly associated with the symptoms of hemorrhoidal disease, e.g. sense of incomplete evacuation, fullness, sphincteralgias, remote gastrointestinal complaints, defecatory inhibition and cathartic habit, etc. This symptomatology together with the chronic infectious processes in the hemorrhoids proper are indications for surgery.

2b. *Contraindications to the injection treatment:*—Those complicating or associated conditions which contraindicate or at least should postpone injection are fistulae, particularly of the submucus variety, acute or chronic indurated fissures

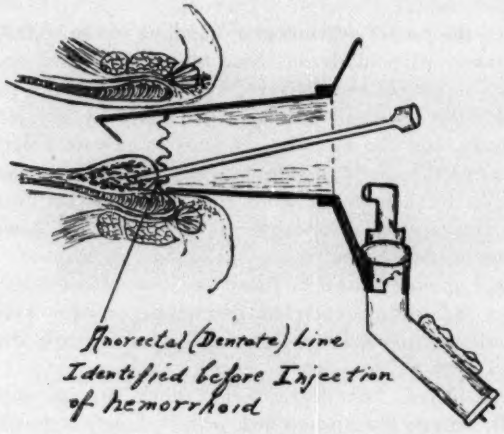


Fig. 13—The low or intrahemorrhoidal injection.

with excessive muscular spasm, chronically inflamed or ulcerated hemorrhoids frequently associated with proctitis, prolapse and pruritis, chronic fibrotic hemorrhoids, large hypertrophic anal papillae frequently accompanied by cryptitis, benign or malignant tumors of the anal canal or lower rectum and particularly hemorrhoids associated with thrombosis of the internal hemorrhoidal veins. A septic thrombophlebitis may well follow the injection of such inflammatory veins. Strangulated hemorrhoids which have been reduced should be injected with caution.

Tuberculous conditions around the anus, the infectious granulomata, amebiasis, advanced hepatitis and nephritis, diabetes and the blood dyscrasias may be additional contraindications.

Technic of Injection

Anatomico-Pathological Considerations:—Obviously one must be familiar with the detailed anatomy of the anal canal and the mucosal patterns covering the hemorrhoid and the sphincteric portion of the rectum above it. The dead line for

the low injection is the anorectal line (dentate, pectinate, etc.) which marks the caudal limits of the internal hemorrhoidal plexus. Its distinguishing points are the valves, crypts, papillae when present and the bases of the columns of Morgagni. With prolapse, the dentate line may be lower than normal. It may be useful to point out that the dentate line may be more readily observed if the anoscope is partly withdrawn and then gently readvanced with slight pressure against the lateral wall of the anal canal. This relaxes the anoderm and makes the edges of the valves prominent.

It may be noted that there is a somewhat abrupt change in the color and vascularity of the mucosa covering the hemorrhoid in contrast to that of the rectal mucosa above it. That covering the internal hemorrhoidal annulus is usually violaceous or plum colored and the veins become less prominent as the upper end of the internal hemorrhoidal annulus is reached. The rectal mucosa above the

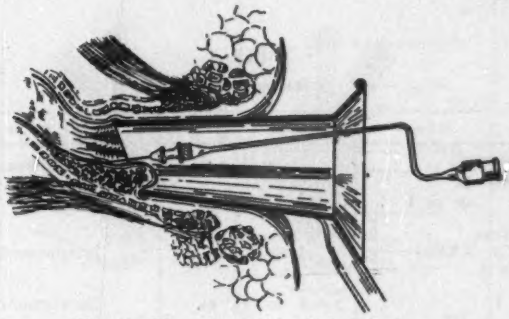


Fig. 14—The submucus or high injection.

hemorrhoid has a uniform brick or pinkish red color and the vascular pattern is quite different than that of the hemorrhoidal annulus.

Pathological changes may obscure the landmarks. In some cases the mucosa overlying the hemorrhoid is replaced by an ingrowth of squamous epithelium, so-called "squamatized hemorrhoids". This is commonly observed in the chronic prolapsing variety with fibrous changes, as the "white hemorrhoid". The dentate line in these cases is apt to be somewhat obscured. Prolapse which may also override the landmarks should be evaluated*.

Armamentarium:—The type of anoscope or proctoscope is of less importance than experience in its use. However, good illumination is absolutely essential and I prefer my own revolving anoscope, with fixed illumination in the handle. This instrument obviates repeated reinsertion both for examination and injection (Fig. 11).

The syringes preferred are 3 and 5 c.c. Luer-Lok type with ringed handles. The needles should be of sufficient length to reach above the anorectal line.

*Those interested in further anatomical details are referred to the author's book: "Perineopelvic Anatomy" for the Proctologist.

Straight, curved or offset needles may be used. We prefer straight or offset needles of 20 G for oil, and 23 or 24 G for aqueous solutions (Fig. 12).

Small alligator forceps, Allis or other nontraumatizing clamps for traction on prolapsing mucosa or hemorrhoids, completes the armamentarium.

Solutions:—Many types of sclerosing solutions are regularly introduced with vaunted superiority over those in use. However, phenol, usually in 5 per cent solution and 5 per cent quinine and urea are still the most universally used and approved solutions. Phenol should be diluted in an organic oil, preferably sweet almond. An inorganic oil should never be used. Quinine and urea is preferably used from ampoules. Phenol in stronger solutions 10 to 25 per cent in water and glycerine appears to be occasionally useful. In an aqueous-glycerine solution it presumably produces a more intense inflammatory and exudative reaction with possibly more terminal fibrosis. It is indicated in overly vascular hemorrhoids, but should be used cautiously. Intolerant dosage may result in severe sloughing with hemorrhage.

TABLE I

SCLEROSANT GUIDE

Sclerosant	*Amount used*	*Site of Injection*
Phenol liq. MXXIV Menthol Gr. II Oleum Amyg. qs oz I	2 to 5 c.c.	Submucus-high
Quinine and Urea Hydro. Gr. XXIIss Aq. des. Oz I	1 to 2 c.c.	Intrahemorrhoidally
Phenol oz I Glycerinum oz III Aqua dist. oz IV	8 to 15 m.	Intrahemorrhoidally
Quinuride	1 to 1½	Optional

Quinuride, a 5 per cent quinine and urea solution with an increased pH to 2.8 recently introduced by Terrell is considered somewhat superior to ordinary 5 per cent quinine and urea. Acids per se have a sclerosing effect on tissues and their addition to the time honored quinine and urea solution would appear to be advantageous. To date our results with quinuride have been very satisfactory. Terrell advises repeated injection with quinuride until maximum shrinkage of the hemorrhoid has been secured.

Highest percentage of phenol now used is 25 per cent as contained in Shuford's solution.

Aqueous solutions have the advantage of better diffusion and less tendency to leak from the needle puncture.

Technic of injection:—Although no special preparation is needed, the rectum should preferably be empty or at least devoid of a large fecal mass which may provoke an undesirable sense of fullness following the injection. The left lateral position, which affords easy relaxation, is preferred. The knee-chest or inverted

position, however, is decidedly preferable in patients with anal prolapse which may obscure the landmarks. Obese individuals with sharp sacral curve are also preferably injected in the knee-chest posture.

Injection of hemorrhoids is usually described with reference to the dentate, anorectal, pectinate, etc., line. However, there are two technics. A low injection or intrahemorrhoidal and a high or submucus injection. The exact points of introducing the hemorrhoidal needle and the distribution of the sclerosant solution are very important. Although satisfactory results appear to follow either method, a combination of the two may give more permanent relief particularly in vascular hemorrhoids with some degree of prolapse. The same sclerosing solution may be used for either injection but we prefer 5 per cent phenol for the submucus injec-

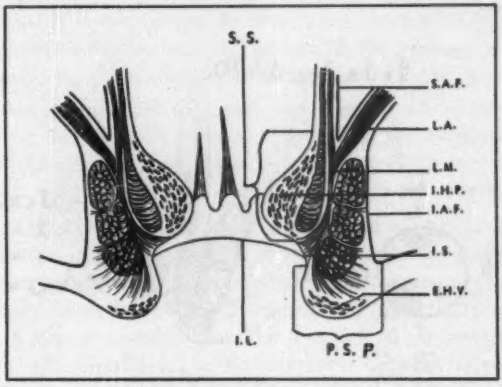


Fig. 15—The submucus and perianal spaces. In the submucus space the anorectal line, the annulus hemorrhoidalis, and its internal hemorrhoidal plexus are the important structures; in the perianal space the terminations of the conjoined longitudinal muscle and the inferior or external hemorrhoidal plexus of veins are to be noted. The intersphincteric line separates the spaces. (S.A.F.) Supraanal fascia; (L.A.) levator ani; (L.M.) longitudinal muscle; (I.A.F.) infraanal fascia; (I.S.) intermuscular septum; (I.H.P.) internal hemorrhoidal plexus; (E.H.V.) external hemorrhoidal veins; (P.S.P.) perianal space; (I.L.) intersphincteric line; (S.S.) submucus space.

tion and 5 per cent quinine and urea, quinuride or 10 per cent phenol in water and glycerin for the low injection.

The low injection is sometimes considered as the "wrong" technic. However, in the author's experience he has found the low or intrahemorrhoidal injection quite useful for hemorrhoids in which the higher submucus injection has failed to control the bleeding or/and prolapse. The low injection is an intrahemorrhoidal injection, usually into the lower end or centre of the hemorrhoid (Fig. 13). This injection calls for absolute identification of the anorectal line. The preferable solution is aqeuous 5 per cent quinine and urea or quinuride or 10 per cent phenol in glycerine and water. The needle should be inserted well into the center of the hemorrhoid. The amount of solution injected varies from 1 to 2 c.c. of the quinine preparations and 2 and 4 c.c. of the 5 per cent phenol solution. Of the 10 per cent

phenol solution no more than 8 minims should be injected and of the 20 or 25 per cent, no more than 5 minims.

The high or submucus injection is an injection into or just above the upper end of the hemorrhoid into the submucus space (Fig. 14). The level of this injection corresponds to the level of the anorectal muscle ring. It may be useful to note that the internal hemorrhoidal plexus, the annulus hemorrhoidalis lies in the submucus space bounded below by the anorectal or dentate line and above by the anorectal muscle ring (Fig. 15).

In the high injection, the anorectal line is first identified and then the hemorrhoid proper and finally its junction with the paler or lighter rectal mucosa. This is the point for the high injection. The needle reaches the submucus space at the extreme upper end of the hemorrhoid and from 2 to 5 c.c. of 5 per cent phenol in oil are injected.

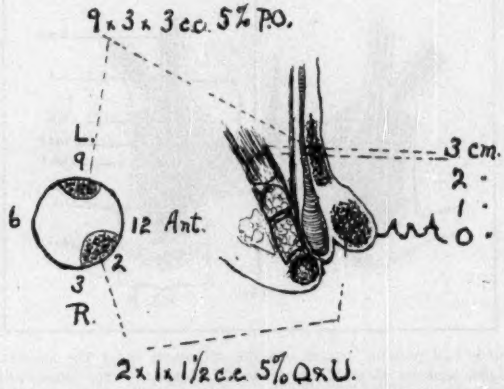


Fig. 16—A simple method of recording the location, level above the dentate line in cm. and the amount in c.c. 12 is the anterior commissure with the patient in the left lateral position, the usual one for injection. The distance between the dentate line and the anorectal ring is about 3 cm. A low or intrahemorrhoidal injection into the right anterior hemorrhoid with 1 c.c. of quinine and urea hydrochloride at about the 1 cm. level is recorded as indicated above. The submucus injection into the left lateral hemorrhoid with 5 per cent in oil at the anorectal ring level is similarly designated.

This high injection tends to correct the prolapse commonly associated with internal hemorrhoids. One will frequently observe that after a large submucus injection of the 5 per cent phenol solution the hemorrhoidal tumors will atrophy to a considerable degree. The amount injected, usually from 3 to 5 c.c. of 5 per cent phenol solution, depends on the extent of the prolapse, the size of the hemorrhoid and the capacity or tolerance of the submucus space at the injected level. This injection into the submucus space is somewhat more exacting than the low injection. It is highly important that the needle be exactly in the submucus space, the needle point must not be fixed either in the mucosa or in the underlying muscle. If the needle is properly placed, the oil flows in easily with the gradual formation of a pale or blanched tumescence which obliterates the smaller capil-

laries but by comparison accentuates the larger ones—"the striation sign". An injection into the mucosa proper produces an immediate totally avascular white blob which may necrose and slough. *The obstructive vascular changes taking place, gradual edema and swelling, blanching, and arteriolar striations at the level of the injection should be most carefully observed.* They are highly significant to the tolerance of the tissues, to the anticipated sclerosis and to any further injections. It may be emphasized that initially large injections give much better results than repeated smaller ones.

The quadrant containing the larger actively bleeding hemorrhoids should be injected first, usually the left lateral containing the primary hemorrhoid. The injection therapy similar to the surgery should reach the vascular pedicles concerned in the formation of the primary hemorrhoids. If they are still prominent and remain vascular it is our custom to then inject them with either a 5 per cent quinine and urea hydrochloride solution or one of the stronger phenol solutions. This injection should be given directly into the center of the hemorrhoid.

The frequency of the injection and the number of hemorrhoids injected depends roughly on the patient's physique the size of the hemorrhoids, the progress in control of bleeding and prolapse and the extent of the fibrosis following previous injections which is determined by careful digital examination.

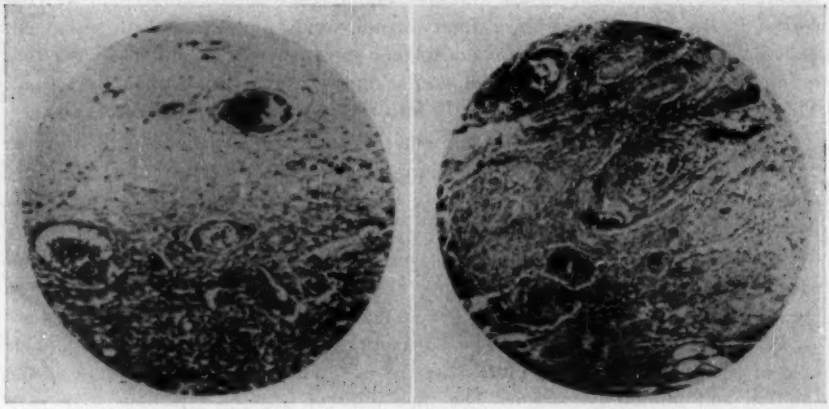
The introduction of the needle in either injection should be painless. Should pain be complained of it usually signifies that the needle has been introduced too low or that some of the solution has gravitated below the dentate line which should always be positively identified before attempting to inject. The needle should be held in place for a minute or more after the injection to prevent the escape of the solution and minimize bleeding. The time interval between injections may vary from 5 to 7 days or more.

The high injections are well above the sensorium of the anal canal. Edema, fullness or other dysasthesias are usually imperceptible and apt to be less troublesome than after the low injection. Deep sloughing with hemorrhage, however, may be more difficult to control. Caution should be exercised in giving injections in the median lines or commissures, 12 and 6 o'clock, the patient in the left lateral position. Careless injection into the rectocele for a supposed hemorrhoid usually referred to as the "perineal pile" has resulted in a rectovaginal fistula. Injections likewise into the posterior prostatic space or the capsule of the prostate have painful sequelae and the injected oil has been passed per urethram.

The aftercare of the injected patients is of some importance. They should be warned concerning the possibility of prolapse of the injected hemorrhoids and we usually instruct our patients to advise us immediately of this complication. An analgesic ointment may be used if desired. If the stools tend to become hard, the instillation of oil at night or mineral oil by mouth are useful. Slight prexia sometimes follows injection but requires no treatment except reassurance of the patient. The occasional case with pain or marked feeling of fullness in the rectum usually yields readily to hot sitz baths and mild sedation.

The injection method should not be used as a substitute for a properly performed surgical hemorrhoidectomy when this is indicated. In properly selected cases, and in these only, the results are very satisfactory and the complications, if the proper technic is used, are practically nil. Recurrences are somewhat more common than with surgery.

Record of Injections:—The simplest method for personal recording is to consider the commissures as 12 and 6 with the patient in the left lateral position. The anorectal line is zero; the level of injection above it is designated in centimeters. The site of injection on the clock, its level and the amount and kind of sclerosant are simply written as shown in Fig. 16.



a. b.

Fig. 17—a. Microphotograph of an internal hemorrhoid injected sixty hours before removal with 5 per cent quinine and urea hydrochloride. Note the marked exudative and cellular type of inflammatory reaction in contrast to phenol 12 per cent, in b.
b. Microphotograph of an internal hemorroid injected sixty hours before removal with 12 per cent phenol solution. Note the more marked thrombosis, and the greater cellular response leading to a firmer fibrosis. Compare with a. Both a and b are from the same patient, the injections being on opposite sides.

If desired the quadrant can be designated and the figures used for distances above the anorectal line and the amount and kind of solution used, e.g. RAQ + 3 + 4 PO. + 5 per cent = right anterior quadrant, 3 cm. above the dentate line, 4 c.c. of 5 per cent phenol in oil.

The unsatisfactory results and complications following the injection treatment have usually followed a poor selection of the cases rather than gross errors in technic or the media used for injection. It may be useful to point out that the injection of a sclerosing solution results in an inflammatory reaction in the hemorrhoidal tissues which presumably produces sufficient residual fibrosis to reduce prolapse and bleeding (Fig. 17). The etiological factors of the hemorrhoidal disease are however, not "cured". One must, therefore, be conversant with the limitations of the injection treatment and the scope of its application.

Complications:—With the proper technic and solutions complications should be of infrequent occurrence. Pain, discomfort and fullness is common following the low injection with seepage into the sentitive anoderm. These usually subside the following day. Immediate hemorrhage from needle puncture of an artery may occur and may entail clamp and ligature. A submucus hematoma with infection abscess, thrombophlebitis and submucus fistula are additional rare complications.

Superficial sloughing and ulceration is fairly common. It is due to faulty technic and intolerant amounts of sclerosants. Chronic anal ulceration may be a troublesome sequel.

Oil tumors were occasionally reported following the injection of inorganic oils in the older technics. They are now less frequent. An inorganic oil should never be used.

Acute quinine reactions in sensitized individuals or those having an idiosyncracy to the drug may be alarming. In suspected individuals, previous test with oral administration of quinine may be informative. Although these acute allergic reactions are comparatively rare, it appears advisable to have immediately available adrenalin and benadryl for parenteral use. Reactions to phenol are extremely rare.

Injection below the skin, into the external hemorrhoidal plexus or into the anoderm or tissues below the anorectal line produces a highly painful edematous, thrombophlebitic mass which persists for weeks (Fig. 18).

Prolapse with partial or complete strangulation is an occasional complication.

Conclusions:—In the last decade innumerable contributions have been made to this subject. Many of these have advocated new sclerosants which appear to have no advantages over those used by experienced proctologists for many years. The only new sclerosant which appears to have some advantage over the common 5 per cent quinine and urea solution is quinuride already referred to and which requires further evaluation.

In summation, the following conclusions may be made:

The injection treatment of internal hemorrhoids is a valuable palliative if not curable method in the properly selected case. It at least may provide a symptomless course. The proper selection of cases requires experience, good judgment with adequate evaluation of the entire hemorrhoidal pathology local and remote. The preferred media for injection are 5 per cent phenol in sweet almond oil, 5 per cent quinine and urea and quinuride. The injection technic is not as simple as usually considered in that it requires a knowledge of the anorectal anatomy. Longer asymptomatic results are to be secured by a combination of the high or submucus followed by a low or intrahemorrhoidal injection repeated as indicated.

End Results:—Statistics concerning "cure" in hemorrhoidal disease following injection treatment are of little or no value because the conclusions are based on criteria subject to wide individual interpretation.

Injections into a slightly varicosed internal annulus are highly curative and usually uneventful while an operation on the same case may also "cure" the patient for any further hemorrhoidal surgery.

Surgical Treatment of Internal Hemorrhoids

Hemorrhoidectomy is commonly considered a simple and minor surgical procedure. The number of patients, however, observed by the author with stenosis of the anal canal, "frozen" anal musculature, painful evacuations and in some cases a persistent rectal neurosis would tend to belie the simplicity of this very common operation.

Moreover a review of the proceedings of the American Proctologic Society for the last 40 years, not to mention the general surgical literature, reveals a variety of diverse and conflicting technical procedures which defy both enumeration and description. They indicate that the functional results even in expert hands is far from satisfactory; new and "composite" hemorrhoid operations have been repeatedly and successively advanced as improvements over but recently advo-

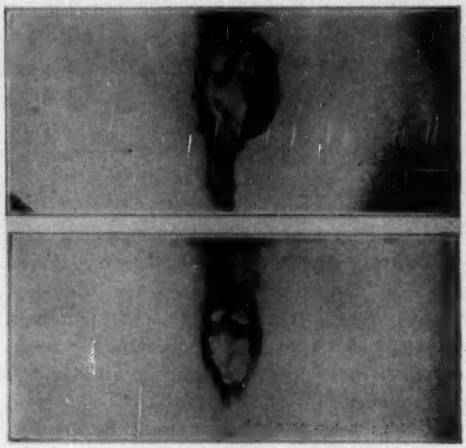


Fig. 18—The upper picture shows the marked perianal edema from an injection of phenol below the dentate line. This picture was taken one week after the injection. The lower picture was taken three weeks after the injection. It was over two months before this cleared up. (*Courtesy of Dr. A. J. Abeloff*).

cated new and improved technics and so on. The latest improvement over the improved technics is the so-called rectoplastic hemorrhoidectomy.

The main reasons for this dissatisfaction may be discussed under the following headings: 1. The selection of cases, 2. Pathological considerations, 3. Surgico-anatomic considerations and 4. Inadequate postoperative care.

1. In the following cases surgery may prove unsatisfactory: Hemorrhoids arising during labor or immediately postpartum are usually recessive and are preferably treated along palliative lines.

Certain individuals, particularly those having experienced previous unsuccesful anorectal surgery are usually unsuitable for radical hemorrhoidectomy. These patients are commonly referred to as "neurotics" (anxiety neurosis), spas-

mophiliacs or the "spastic type". It appears that anal scarring with more or less persistent postoperative spasm of the anorectal musculature provokes a variety of chronic complaints. A permanent hypochondriasis, rectal neurosis or cancer phobia may supervene. Surgery should be the last experimental therapy in these patients.

Finally in patients with active proctocolonic infections, chronic ulcerative colitis, active amebiasis, lymphopathia venereum or other active venereal disease surgery is at least temporarily contraindicated. Hemorrhoidal surgery in the presence of readily palpable or visible cancer is a sad commentary on inadequate proctologic examination.

2. *Pathological Considerations:*—Associated pathological conditions in hemorrhoidal disease require careful evaluation in the surgery. Unsatisfactory results in hemorrhoidectomy frequently arise from a failure to properly evaluate and correct associated pathology or that which may be of etiological significance. For example, failure to recognize and completely incise the anal fibrosis or a pecten band in hemorrhoidectomy may result in severe postoperative dyschesia and dyskinesia, since the trauma and infection of the operation itself sometimes also unduly constricts the anal orifice. Frozen musculature with excessive defecatory straining and reflex gastrointestinal symptoms may supervene.

"Cryptitis", papillitis, "pectenitis", anal fibrosis, excessive sphincter spasm, anal ulcerations, submucus fistulae and particularly fissure are basically infectious processes significant to the symptomatology and etiology of hemorrhoids. The correction of these conditions may be of more importance that the removal of hemorrhoids or supposed hemorrhoids. Deep and frankly infected crypts should be drained. However, too much surgical enthusiasm should not be expended on crypts. We would again emphasize that it is not so much the crypt but the pecten tissues, glands, ducts, etc., which harbor the infected foci.

The recesses of the crypts, moreover, do not drain into the submucus space which contains the hemorrhoids.

A complicating chronic anal fissure requires complete division of the subcutaneous external sphincter muscle and the anal fibrosis with adequate skin drainage.

3. *Surgicoanatomical Considerations:*—Hemorrhoidectomy with all its variations over the ages has been one of the commonest of surgical procedures performed with little or no regard for the anatomy. Moreover, the correct anatomical basis for the bulk of anorectal as well as colonic surgery is as yet not too well established.

The anatomical relationship of the extensions of the conjoined longitudinal muscle to the anorectal musculature particularly to the subcutaneous external sphincter muscle, and to the hemorrhoidal plexuses are basic to any technic, surgical or otherwise. Additionally, the anal intermuscular septum, the anorectal muscle ring and the subcutaneous external sphincter muscle are landmarks which should be readily identifiable.

Further the composite components of hemorrhoids and their relations must be recognized e.g.—the columnar brick colored mucosa of the rectum above the internal hemorrhoidal plexus, the stratified plum colored striated and more vascularized rectal mucosa overlying the hemorrhoid proper, sometimes referred to as the anal mucosa and finally the skin of the anal canal—the anoderm. The anal canal, unless one considers the sphincteric portion of the lower rectum as anal canal, has no mucosa; there is no anal mucosa.

The detailed anatomy of the anal musculature and anal canal is available in the author's book, "Perineopelvic Anatomy from the Proctologist's Standpoint".

4. *Inadequate Postoperative Care:*—Routine postoperative measures usually suffice. However, of considerable importance is the alleviation of excessive muscu-

Fig. 19—Location of novocaine wheal for injection of Anucaine in hemorrhoidectomy.

lar spasm which interferes with drainage, favors thrombosis, edema, skin "tagging" histaminosis, tissue anoxia, pain, ultimate scarring and stenosis of the anal canal. Scarring in the commissures, particularly the posterior, which may form the basis for fissure may be particularly troublesome and resistant.

Sphincterotomy and the preoperative injection of an oil soluble anesthetic—anucaine, in our hands has favored a smoother convalescence. The preoperative use of the sulfonamides and antibiotics as a routine measure in hemorrhoidectomy to prevent postoperative infections and presumably hasten healing require further study and evaluation. Sulfasuxsidine appears presently to be preferred. In addition to its bacteriocidal effect it forms a soft pultaceous stool which may be indicated in the obese constipated or obstipated patient.

Postoperative dilatation is sometimes important. It should be used as indicated and not routinely.

Hemorrhoidectomy Technics

The number and variety of surgical technics embraced by the term hermorrhoidectomy defy both enumeration and description. In general they include ligature, ligature and excision with or without a clamp, the clamp and cautery, amputative or proctoplastic operations, and several electrosurgical procedures. An infinite number of minor variations are regularly advanced as a new operation with some special suture or clamp.

In general, hemorrhoidectomy may be divided into so-called open and closed methods. Complete mucosal and skin closure or inversion, burying of stumps, proctoplastic closure, etc., are considered inadvisable and unnecessary by the author. They tend to provoke the complications which they are designed to avoid, namely inadequate drainage, infection, edema, thrombosis, pain, sphincter spasm and anal stenosis. They add nothing to what may be accomplished by a simple open hemorrhoidectomy. The observance of fundamental surgical principles is of far more importance in anorectal surgery than technics devised to shorten convalescence and "cosmetize" the perianal skin. Moreover, the routine use of these methods is to be severely condemned. In our experience simple ligature methods to be described here in detail have sufficed for the usual run of internal hemorrhoids. These simple technics may be modified as the particular case indicates. Amputative technics should only be used when the indications are specific and only in experienced hands.

Ligature Operations

Ligature or tieing off with excision of hemorrhoids is steeped in antiquity having been practiced by Hippocrates and Galen. Throughout the centuries it was a crude procedure, complicated by hemorrhage, severe pain, sloughing, pyemia and a not infrequent fatal outcome. But little advance was made in the surgical management of hemorrhoids for some 23 centuries until Salmon in 1833 the founder of St. Marks Hospital, in London modified the hemorrhoid operation by providing adequate drainage of the anoderm and skin below the hemorrhoid which was "separated from its connection with the muscle and submucus tissues upon which it rests". The resultant pedicle was tied at the neck of the tumor, including the vessels. "The superabundant skin was cut off". This operation was used routinely at St. Marks Hospital, London for some 40 years. Sir William Allingham, Salmon's successor, described the Salmon operation in his text book on "Diseases of the Rectum" published in 1888 as "the safest and best operation for the majority of cases of hemorrhoids".

In 1883 the method was introduced into America by Joseph McDowell Mathews the "father of American Proctology". This operation has formed the basis for the large variety of ligature and excision technics, with or without clamps. Ligature and excision technics continue to be the popular operations for internal

hemorrhoids in this country largely supplanting the time honored clamp and cautery and the mutilating Whitehead operation, at least in the hands of the proctologist.

Modifications of the original Salmon operation were introduced primarily to control hemorrhage and to hasten healing by partial closure of the wound with or without a special clamp. One of the more popular modifications was that devised by Hirschman in 1925, and described as his so-called "bloodless ligature operation", which is still one of the more popular ligature and excision technics. It is a simple and surgically sound technic to be subsequently described.

In 1923 Pennington described an open operation in which he dissected out and ligated the vessels in the hemorrhoidal bed after partial excision of the overlying hemorrhoidal mucosa and the adjacent anoderm and skin. This operation required careful hemostasis and its use in large hemorrhoids with prolapse was attended by considerable hemorrhage which led to its general unpopularity.

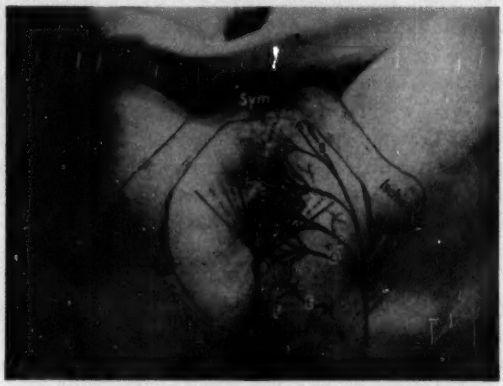


Fig. 20—The distribution of Anucaine in relation to the perineal nerve supply. (A) Perineal nerve; (B) pudendal nerve; (C) anterior sphincterian nerve; (D) inferior hemorrhoidal branch; (E) fourth sacral nerve; (F) coccygeal branches.

The so-called ligature and clamp operation originally accredited to Mitchell of Ireland was popularized in America by Earle who devised a special clamp—the Earle Clamp—for it. Many pros and cons have been expressed concerning the clamp. Its continued popularity has been largely due to its routine use in hemorrhoidectomy by Bacon, Buie, Ross and man yother teaching proctologists.

In 1937 Milligan and Morgan and their cwoorkers devised and described a ligature and excision technic which is presently used routinely at St. Marks Hospital in London, and is becoming increasingly popular both here and abroad. The author considers this operation as the most satisfactory yet devised for the usual run of internal hemorrhoids. In his hands it has been very satisfactory. It is the only hemorrhoidal operation done on a strictly anatomic basis.

The present modifications of the ligature operations are the so-called "proctoplastic" hemorrhoidectomies which require careful evaluation. These innovations appear to complicate a surgical objective attainable by much simpler, safer and sounder methods.

General Considerations

No one hemorrhoidal technic is applicable to all cases and much of the dissatisfaction following hemorrhoidectomies stems from the routine use of one method. However, the following generalizations, concerning hemorrhoidectomy are considered applicable irrespective of the technic.

Regardless of the operative position, lateral Simms, jackknife, lithotomy, etc., and the anesthesia used, adequate exposure is essential to a good technic. Exposure depends on sphincter dilatation, not divulsion, which may or may not be necessary. In cases with pectenosis, pecten bands or anal stenosis exposure should be secured by posterior sphincterotomy without further ado. A patient in whom the anal canal cannot be dilated to at least two fingers under proper anesthesia requires sphincterotomy aside from the dilatation. Without it the operation may lose much of its value. Moreover, the extent of the hemorrhoidal disease with the prolapse may be entirely missed without a sphincterotomy.

Exposure with triangular Pennington clamps or skin clamps is very satisfactory and observing all the hemorrhoidal pathology and the prolapse at one time is conducive to a planned and complete operation. So-called operative specula used and advocated for hemorrhoidectomy are considered unnecessary if not obstructive for the average case. They are occasionally useful in searching for high internal opening of fistulae or transfixing high tumors sometimes associated with hemorrhoids, or suturing plastic skin flaps to the rectal mucosa as in the Fansler or other amputative technic.

Preservation of anorectal physiology is of some importance. Sufficient anoderm must be left between hemorrhoidal stumps or linear areas of excision for adequate regeneration. This is particularly important in the operation for strangulated hemorrhoids. This also applies to the anal skin verge in the amputative type of hemorrhoidectomy and in large mixed hemorrhoids with a collar of skin tags. Excessive scarring and constriction is particularly disabling to the posterior commissure where an intractable fissure may supervene. These sequelae may be avoided by sphincterotomy, adequate skin drainage and good postoperative care. Hemorrhoidal pedicles should not be stripped up widely into the rectum above the intermuscular septum. Severance of the extensions of the conjoined longitudinal muscle in and above the intermuscular septum permits high retraction of the stumps and may leave large denuded areas in the narrowest portion of the anal canal and sphincteric portion of the rectum and incidentally exposes the sensory zone of the anal canal.

Adequate skin drainage is of some importance. Excessive removal of the internal veins without removal of the corresponding external component favors edema, thrombosis, anal spasm, skin tagging and a painful convalescence.

Preoperative Preparation

The preoperative preparation and orders for hemorrhoidectomy depend somewhat on the operative time and the type of anesthesia. Although printed instructions are convenient and time-saving the necessity for specific measures in some cases should not be inadvertently overlooked. For routine anorectal surgery the following preparation may be used:

Rectal preparation:—The perianal skin should be shaved if local infiltration anesthesia is used and also the site of needle punctures shaved before injecting oil anesthetics.

Catharsis:—No catharsis is used except in cases not having bowel movement for several days. Castor oil in appropriate dosage is used unless there are specific contraindications. An enema is given routinely from 4 to 6 hours preoperatively.

Diet:—There are no restrictions except to foods to which patients have known allergy or other idiosyncrasy.

Sedation:—Barbiturates in appropriate dosage are given the night before operation and repeated if necessary in 3 hours for sleep. Barbiturates are again given 3 hours preoperatively in dosage of gr. 1½. Narcotics are given about one hour, preoperatively in indicated dosage, we prefer demoral 100 mg. with atropine gr. 1/100. Small doses of hyoscine are occasionally added to this.

Chemotherapy may be useful in the stocky gaseous constipated type of patient. Cases with strangulation and active infectious processes may require additional antibiotic therapy. In patients with anemia, liver, folic acid and iron injections may be used several weeks before surgery. If the hemoglobin is below 60 per cent transfusion should be considered.

Anesthesia

A routine anesthesia should not be used, the preferred anesthesia at least by the proctologist for anorectal surgery is spinal, followed by pentothal, caudal with or without transsacral. Inhalation anesthetics and local infiltration notwithstanding their undesirable complications are still used. We prefer spinal with a maximum of 50 mg. of procaine (through a needle of 26 gauge) dissolved in the spinal fluid unless contraindicated. We combine the local injection of the oil soluble anesthetic Anucaine originally introduced by us in 1928. For caudal we prefer 30 to 40 c.c. of 1½ per cent procaine in saline without adrenalin followed by 30 c.c. of normal saline to spread the novocaine. For transsacral we prefer 2 per cent novocaine in saline with the usual indicated dosage for the respective foramina.

If pentothal is used we prefer the lithotomy position. In the aged with relaxed musculature local infiltration of procaine may be indicated.

Headaches:—Low occipital or frontal headaches are a fairly common and troublesome complication following spinal anesthesia. It is probably due to an intermittent or continuous leakage of spinal fluid. Its prophylaxis consists in using the smallest spinal needle, 26 gauge, and if possible only puncturing the dura once. Active treatment consists in the caudal injection of 30 to 60 c.c. of normal saline which presumably neutralizes further leakage by increasing the fluid tension in the

tissues adjacent to the site of dural puncture. Persistent headaches may require direct intraspinal injection of 30 to 60 c.c. of normal saline solution to re-establish the subarachnoid pressure. Intravenous glucose-saline, vitamin injections and the use of analgesic drugs may be of some placebic value.

Injection of Anucaine in Hemorrhoidectomy

This oil soluble anesthetic is used to prolong relaxation of the sphincters and to decrease postoperative pain and catheterization. It is not injected primarily for the operative anesthesia. This has been a very common mistake in using oil-soluble anesthetics. Anucaine is used regardless of the anesthesia, spinal, caudal, pentathol, etc., and it is occasionally combined with local procaine or penicillin-procaine in indicated cases.

The technic of the injection consists in distributing 5 c.c. of Anucaine into the posterolateral quadrants of the perianal region primarily to reach the main branches of the inferior hemorrhoidal nerves as they traverse the ischiorectal space to reach the sphincters. The sphincters are usually not injected. If local anesthesia is used an 18 gauge needle is inserted in the midline through a novocaine skin wheal, placed about 3 or 4 cm. posterior to the anal verge (Fig. 19), and 5 to 8 c.c. of the oil distributed as evenly as possible on each side (Fig. 20).

Anterior injection 3 to 5 c.c. are then made through the lateral aspect of the perianal skin to reach the anterior branches of the inferior hemorrhoidal nerve as well as the anterior sphincterian nerve. This affords a complete perianal injection. The tissues are then firmly massaged to further spread the oil.

Anucaine should preferably be used *before* any operative procedures are started.

(*To be continued*)

VARICES OF THE ESOPHAGUS*

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The presence of varices of the esophagus insures a serious clinical problem. The condition follows an obstruction of the portal blood supply and its existence is made known in an acute form whose main feature is announced in a greater or lesser blood loss, or it can be a manifestation of a chronic pathological process with or without accompanying symptoms. The relative infrequency of varices does not bespeak its diminished importance and need for serious consideration. An unexplained secondary anemia, varying degrees of hemetemesis or melena calls for consideration of this condition as a possible etiological factor. Indeed, not unusual are the occasions when it requires differentiation from an ulcer state in which massive hemorrhage takes place. The presence of an enlarged liver or spleen, an accumulation of abdominal fluid or the recognition of engorged and visible abdominal veins gives impetus to the possibility of varices of the esophagus. The intake of large quantities of alcohol has been regarded as a precursor of a cirrhotic liver, with an attendant impaired food intake as the actual offender in the production of liver damage. Other times, the history may contain evidence of some toxic basis, and less frequently, a thrombosis of the hepatic vein or the local pressure of enlarged glands may initiate a circulatory blockage. Extrahepatic causes likewise play a part in the causation of these enlarged veins, such as a thrombophlebitis of the venous system following trauma or infection as well as a congenital obliteration of the portal vein. It is possible for a combination of both intrahepatic and extrahepatic factors to exist simultaneously. Whipple[1] reported a cavernomatous transformation of the portal vein as causing an extrahepatic block. It may be noted that an impairment of the portal blood supply may take place in a liver which is normal in size, as well as one which may vary in either direction. The mechanism of portal obstruction has been understood for many years. The known hypertension of the portal, gastric and splenic veins has intrigued the minds of investigators and has stimulated research in the understanding of the alterations of structure and function. Eck[2], in 1877, demonstrated experimentally on animals that portal venous blood could be shunted directly into the stystemic venous network by anastamosing the portal vein directly to the inferior vena cava thereby by-passing the liver. This thought was the foundation of future surgical ideas which soon became directed into procedures of a practical nature.

It need not be emphasized that the diagnosis of varices is of vital importance. It has been stated that roughly 20 per cent of patients affected with cirrhosis of the liver die of hemorrhage. When the degree of venous enlargement becomes such that hemorrhage ensues, the problem immediately becomes formidable, for there is no easy road from then on. In the early stages of cirrhosis, varices are

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not likely to be present, but if it were possible to determine its existence in its incipiency, it might well be that the approach to therapeusis or surgical attack might assume an entirely different aspect. The calamatous state of an exhausting hemorrhage scarcely offers a favorable opportunity for optimism, not to speak of hope.

A sudden loss of blood may be the first sign of an esophageal varix. On physical examination, an enlarged liver or spleen may be felt as previously mentioned. There may be an anemia of varying extent, a low platelet count, retention of bromsulfalein, a reversal of the albumin/globulin ratio, reduced serum albumin, positive cephalin flocculation test, and an elevated prothrombin time. Some jaundice may be apparent. In the presence of an extrahepatic lesion, these tests may be entirely negative. Since time immemorial the existence of gastric and esophageal varices has been recognized at the postmortem table. The roentgen demonstration of these enlarged veins did not take place however until 1928[3]. Its introduction at that time added a valuable means in diagnosis. Unfortunately, through its use, early recognition of venous enlargement is not possible. Furthermore great skill and a special technic are required by the examiner, and during active hemorrhage, the roentgen method of examination cannot be resorted to. However, it is by all means advisable that in all suspected cases of varices, unless a contraindication is apparent, that a roentgen attempt at diagnosis be invoked. It might be noted that varices of the gullet often extend down into the stomach. The latter veins may be large enough to simulate a tumor of the cardia or fundus[4].

The esophagoscope affords a means for accurate diagnosis. In the early stages when the veins are likely to be small, other measures may fail, but by direct visual inspection diagnosis is accurate and reliable. In the presence of actual bleeding, instrumentation may be the only direct method by which the true nature of the existing condition may be made known. Especially, since the introduction of flexible tube esophagoscopy[5], the fears and restraints in this form of examination are no longer justifiable. In suspected cases, long before actual hemorrhage has taken place, the presence or absence of varices, if determinable, can only add to the efforts of the clinician and surgeon. It is surprising how frequently varices are thus observed which would otherwise be overlooked, and how often, where expected, one finds a perfectly normal esophageal mucosa.

It is common knowledge that all varices of necessity do not bleed. Rivers[6] et al. estimated that about 5 per cent of 668 cases of cirrhosis or Banti's disease experienced hematemesis. Costello's[7] figure yielded an 8 per cent incidence of ruptured varices in 300 cases studied by him. It is Wangensteen's[8] belief that hemorrhage from the esophageal veins can be traced to peptic ulceration of the esophageal mucosa covering these veins in consequence of the acid reflux from the gastric juice.

The approach to treatment depends obviously on the extent of the problem at hand. An early diagnosis may be a blessing to the patient who can be forewarned of the consequences of neglect or the advancement of the disease process. Much can be accomplished in this way in anticipating disaster. Certainly, when

dealing with varices, prevention is much more hopeful than cure. At the present time the treatment of varices of the esophagus is purely palliative and an emergency hemorrhage may tax one's resourcefulness to the utmost. A profuse loss of blood may thus defy the limited means at one's disposal. Westphal[9] in 1930 resorted to the use of tamponade by employing a Gottstein sound. Other observers[10] since have had recourse to similar methods. Dolowitz and his co-workers reported the control of severe bleeding from esophageal varices with the use of oxycel[11] applied locally to the bleeding point through an esophagoscope. The speaker himself has had satisfaction in the use of gelfoam under similar circumstances.

In 1939 Crafoord[12] introduced the injection treatment of varicosities and thus was able to control hemorrhage for certain periods of time. However, one cannot be assured that recurrence of bleeding would not take place. A patient suitable for injection therapy would be one who had experienced a recent hemorrhage, but who does not present evidence of extension of the enlarged veins into the stomach. In the latter event, the likelihood of success is rather remote. It is a further observation that as the esophageal veins shrink after treatment, the gastric veins become enlarged to the degree that subsequent bleeding from this source may be added to that of the esophagus. One may therefore not depend too forcefully upon the injection method as an answer to one's need in this difficult problem.

In the 19th and 20th centuries, various shunt operations were performed. Few however, were successful, and so this form of approach was finally abandoned. Whipple and Blakemore[13] both reported successful examples of portocaval shunt as a means of diverting the blood flow. These authors were the first to describe the splenorenal shunt operation. Phemister[14] recommends removal of the bleeding segment by means of an esophagogastric resection. This procedure may be indicated where bleeding arises mostly from the stomach itself. Resection in this way parallels the injection method of treatment by throwing more load on the collateral circulation. This may be justifiable because the patient is apt to bleed less often and possibly not as intensely. In bleeding in Banti's disease, a splenectomy is advisable and possibly an omentopexy to help establish a collateral circulation between the portal and caval systems. It is worthy of emphasis that early removal of the spleen in Banti's disease is desirable before the development of gastric and esophageal varices takes place. A portocaval shunt in Banti's added to a splenectomy has aided in the relief of the passively congested organs drained by the portal system. Recently, Som and Garlock[15] advocated a posterior mediastinotomy for the purpose of shunting the blood flow into deeper channels.

Despite these various surgical procedures, the danger of recurrent hemorrhage still persists. It appears therefore that prevention if possible of the underlying causative condition, and a more detailed diagnostic awareness of existing pathology as only esophagoscopic instrumentation can insure is more likely to lead us out of perplexity and uncertainty into possible hope and accomplishment.

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GIANT ECHINOCOCCUS CYST OF THE SPLEEN*

Case Report

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This patient was an Italian male of 65 years who had been brought up in Italy and who had returned to Italy several times since then. He had an enormous echinococcus cyst originating in the spleen. The diagnosis of splenomegaly had been made before operation.

Before describing the case history, it might be advisable to review briefly echinococcus or hydatid disease.

The adult form of the parasite lives in the small intestine of the dog or wolf, and the larval stage occurs in man and some domesticated animals. Man is infected by swallowing the eggs of the adult worm discharged in the feces of a dog. Escaping from the intestines, the embryo may migrate to any part of the body, but most frequently it enters the liver. The embryo once at rest slowly develops into a hydatid or echinococcus cyst. The latter consists of an outer thick, translucent, laminated membrane—the ectocyst—and an inner, thin, granular, germinal layer —the endocyst. From the endocyst originate small buds, which become brood capsules of immature heads of the adult parasite, called scolices. If the wall of the brood capsule is ruptured, the scolices may become detached and float free with a specific gravity of 1.004-1.014. As the parent cyst grows, daughter cysts and even granddaughter cysts containing scolices, with their characteristic hooklets, may form and later become free in the hydatid fluid. In this way the original cyst may attain a weight of many pounds and contain scores or even hundreds of daughter cysts. As a result of spontaneous rupture or evacuative puncture of a primary hydatid cyst, scolices may be set free and give rise to the formation of secondary cysts in the surrounding tissues or organs.

In rare instances, probably through infection by a distinct species of parasite, the echinococcus multilocularis, there is formed the so-called multilocular cyst which is a tumor-like mass consisting of numerous small cavities filled with gelatinous fluid and usually communicating with one another.

Cysts may occur in any part of the body, even in the heart, the bones and the eye, but the liver is involved in two-thirds of the cases, the lungs in 10 per cent of the cases and the kidneys in about 8 per cent. The spleen is rarely involved, only in about 2 per cent.

Through the courtesy of Dr. Lisa, I report the following case: "L.B.—White male, 53, Italian, under observation for diabetes and peripheral vascular disease.

"At autopsy there was found a very large spleen with two large intercommunicating sacs. The larger sac had a calcified wall and small masses projecting from its inner surface. The smaller sac had a fibrous wall. The contents were brown-yellow.

*Presented before the Clinical Meeting of the New York Chapter of the National Gastroenterological Association, New York, N. Y., 9 April 1951.

"*Diagnosis:*—Echinococcus cyst of spleen."

I now quote from A. Jentzer's article in the *Schweizerische medizinische Wochenschrift,* January 11, 1947:

"Echinococci cysts of the spleen are rather rare; cases have been observed between the ages of 10 and 74 but the age between 25 and 35 is most frequently involved. In Switzerland over a period of 27 years, 102 cases of alveolar and 100 of hydatid echinococcus have been reported; of the former, 95 involved the liver, two the brain, and one each the lungs, five the spleen, two each the omentum and the thyroid gland, and one each the kidney, ovary, parametrium and the abdominal wall. Curiously the majority of cases of alveolar echinococcosis were found in the German and that of hydatid echinococcosis in the French portion of Switzerland; zoologists are not certain whether two different parasites are dealt with or whether the alveolar echinococcus is a degenerated hydatid one.

"It has been assumed that the embryo may migrate directly from the intestine to the vena cava inferior through the lower and middle hemorrhoidal veins without passing through the liver, or may penetrate through the intestinal mucosa and migrate through the chyliferous ducts and the thoracic duct into the vena cava superior and the general circulation; some authors thought that under the influence of abdominal strain the embryo may migrate from the portal vein into the splenic one and thus reach the spleen more readily as this venous system has no valves. Finally one author reported venous anomalies, with some veins from the left colonic angle and part of the descending colon extending directly to the lower pole of the spleen. The classic theory still seems most likely; according to it the embyro would perforate the intestinal mucosa, penetrate into a vein and be carried through the portal vein into the liver."

The case now reported is that of a male, age 65, who gave a history of swelling in the left upper quadrant for the past 30 years. This swelling had gradually gotten worse but at times following x-ray therapy there was some regression.

It is interesting to note that this patient was originally sent to the Memorial Hospital by his family physician for an undiagnosed splenomegaly. X-ray therapy was ordered and a large number of such treatments were given him for over a year with little relief and a resultant anemia and weakness. When first seen by Dr. Seiler in October, 1945, he presented this large tumor mass in the left upper quadrant. It was firm, of a considerable size, filling the entire L.U.Q., causing the ribs to flare outward and causing respiratory embarrassment with poor appetite because of the pressure on the stomach. Patient then received a series of injections of liver and iron and vitamins and he was referred to the Hackensack Hospital for further x-ray therapy. No improvement followed and he was referred to me for possible splenectomy. After examination it was decided that his condition was inoperable. Further x-ray studies were made to rule out any pathology in the G.U. tract.

Several months later we noticed a cystic feel to the mass and because of his marked dyspnea and general distress, it was finally decided to hospitalize the patient and explore the cystic mass. Laboratory reports dated July 7, 1947, were

as follows: Urine: Acid. Sp. gr. 1.019; albumin negative. Microscopic: bacteria. Blood: R.B.C. 3,020,000; W.B.C. 7,750; Hgb. 70 per cent; Polys 57 per cent; small lymph 42 per cent; eosinophiles 1 per cent. Morphology: app. normal.

On May 14, 1947, the patient was operated upon. A small incision was made over the cystic area and a large trochar and cannula were inserted. A large amount of fluid was evacuated but because the trochar could not drain the area, under local anesthesia an incision was made and a finger inserted and two large basins full of daughter cysts were evacuated. A large rubber drain was inserted and the cavity irrigated with tyrothricin solution. Large doses of penicillin were given as well as supportive treatment of glucose solution, high protein diet, liver and iron injections and blood transfusions.

The pathological report from the Paterson General Hospital was as follows:

"Specimen consists of about 3,000 c.c. of pinkish yellow, gelatinous material.

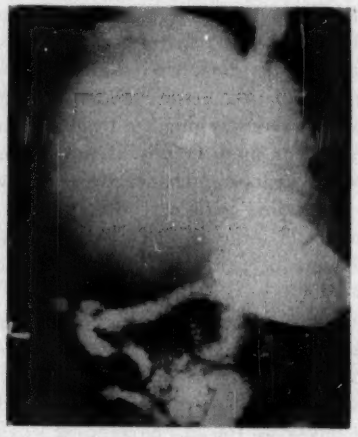

Fig. 1—X-ray showing the large cyst pressing the stomach toward the right.

Throughout this material can be seen numerous thin wall cysts, varying in diameter from a few mm. to 4 cm. These cysts are filled with clear, thin fluid which apparently exhibits only a small amount of mucus. The wall of these cysts measures less than 1 mm. in thickness. Throughout, the entire specimen is thin, white, membranous tissue which apparently represents the wall of a larger cyst. When some of the pieces are reconstructed, they seem to represent preruptured cysts, some of which measure 10-15 cm. in diameter. Very careful search throughout the material fails to reveal any solid tissue other than that representing the wall of intact cysts or previously ruptured cysts.

"The second specimen consists of an irregular, grey to brown, piece of tissue which, on section, is not remarkable.

"*Microscopic:*—Several sections prepared. They all show structureless mucinous material. No epithelial or endothelial cells can be found. Very careful search

of the fluid and cysts was made but no diagnostic features of echinococcus can be found. May I suggest that a skin test be made for echinococcus.

"*Pathological diagnosis:*—Echinococcus cysts or pseudomucinous peritonei. A. H. Davis, M.D., Pathologist."

The drainage and irrigation were continued until July 6, 1947, when the patient was again hospitalized upon his insistence that something be done. The drainage had now assumed a very sour, putrid odor that made everybody keep away from him and he could not stand it himself. The previous mass was now about one-third the original size.

In July, the patient developed a transient diabetes as shown by the following laboratory findings:

7/5/47	B. sugar	— 386	mg. per 100 cc. blood
7/9/47	"	— 197	"
7/12/47	"	— 117	"
7/16/47	"	— 82	"
7/22/47	"	— 110	"
7/25/47	"	— 170	"
7/30/47	"	— 114	"
8/18/47	"	— 82	"

phragm and pushed the stomach toward the median line. The left lobe of the liver was not involved in the mass. It was felt that the spleen was definitely connected with the large echinococcus cyst, so a splenectomy was performed with a

On July 23, 1947, a second operation was performed. A large L-shaped incision was made, enclosing the fistulous tract. A large cavity was entered, which was closely incorporated with a fixed spleen. The cavity extended upward to the diasmall portion of the tail of the pancreas. Large calcified plaques were peeled off the diaphragm and the lateral margin of the stomach. Wound closed with drain to left subphrenic and left lumbar gutter. The operation took about 2½ hours.

The patient had a stormy convalescence but gradually recovered and was discharged on August 18, 1947.

The pathological report on the specimen taken on July 23rd is as follows:

"Specimen consists of numerous pieces of rather dense tissue apparently representing capsule of some type of cyst wall. When laid out, the largest piece measures 15 cm. in diameter. The pieces vary in thickness from 0.2 cm. to about 1.0 cm. Each surface reveals greyish white, necrotic material. It is impossible to recognize definitely which surface represents the inner portion of the cyst wall. In some places the cyst wall contains plaques, apparently representing calcium deposits. There are also several small pieces of fat which contain hemorrhage. One large piece is attached to the entire spleen or portion of spleen. The portion of spleen submitted weighs 170 grams. It is irregular in shape. One surface reveals a dense capsule made up of tissue similar to the other pieces of tissue described as representing the cyst capsule. The portion of splenic capsule measures 0.2 cm. thick. The other surface of the spleen is covered with a similar, but less thickened, capsule. The margins of the spleen are roughened and apparently portions of it

have broken away. The splenic parenchyma, after fixation, is dirty grey brown in color and apparently reveals increase in fibrous tissue. Further sectioning of the spleen also reveals an occasional, well demarcated, yellow area grossly characteristic of infarct, largest focus measures 3 cm. in diameter.

"*Microscopic:*—Sections prepared from the fragments of cyst wall as described grossly show dense hyalinized fibrous tissue with the deposit of a small amount of calcium. No epithelial or endothelial lining can be demonstrated. Sections prepared from the spleen show a markedly thickened capsule and a recent infarct. One section also shows a small piece of pancreas, not remarkable pathologically.

"*Pathological Diagnosis:*—Chronic perisplentitis. Fragments of old cyst wall. Type of cyst not classified. Portion of pancreas, not remarkable pathologically. A.H. Davis, M.D., Pathologist, Paterson General Hospital."

Fig. 2—Lipidol injection showing the remaining cavity after the first operation.

Dr. Lisa's report on the same specimen is as follows: "*Spleen:*—The spleen shows extensive scarring and old hemorrhage. Hooklets are not demonstrable within the spleen but the sedimented fragments of material from the cystic area show many of them.

"*Diagnosis:*—Echinococcus cysts of spleen."

Following the second operation, the patient developed a thrombophlebitis of the right popliteal with severe pain in the leg and foot and cyanosis of the big toe. He received several paravertebral block injections, with recovery.

Now he is well and goes to work. He has gained considerable weight and looks and feels better. His wound has completely healed.

Summary

This patient presents the unusual condition of having had an enlargement in the region of his spleen for thirty years. The diagnosis of splenomegaly had been

made and we did not suspect echinococcus disease at the time of the original operation and therefore had not had any serological tests.

It is interesting to note that he had eosinophiles of only one per cent. During the course of his disease, he developed a transient diabetes which must have been due to the nearness of the septic area to his pancreas, because since operation, he has had no signs of diabetes.

The final excision of his cyst was a procedure of considerable difficulty because of the marked calcified plaques lining his diaphragm, stomach and lesser peritoneal cavity. With no focus in the left lobe of the liver and with a spleen innately adherent and part of the cystic wall, one must assume that primarily this was echinococcus disease of the spleen.

GASTRIC RESECTION FOLLOWED BY STRICTURE OF THE COMMON DUCT*

CASE REPORT

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A patient with a history of a number of years of duodenal ulcer, had an acute flare-up two weeks before I saw him. The original x-rays showed duodenal ulcer with small perforation and abscess cavity. Following subtotal gastrectomy, he developed jaundice and had four more admissions to hospitals for treatment of jaundice and chills and two operations, with eventual recovery.

Mr. A.T., age 44, consulted me on April 18, 1949 complaining of epigastric pain, distention and vomiting. He was a singer by profession and had always been very athletic. His main history is as follows:

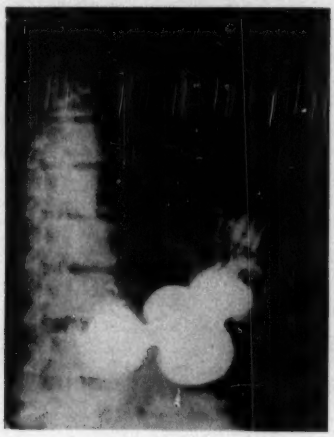


Fig. 1—Gastric x-ray before first operation, showing penetrating ulcer at the top of duodenal cap.

He had had a duodenal ulcer for 13 years; had tried a rather inadequate dietary regime. In the interim between attacks, he could eat anything. Two weeks before I saw him, he felt food stick in his stomach and had a constant gnawing epigastric pain. After eating, he felt distended and caused himself to vomit. Two months previously, he had been on a diet and had lost 30 pounds.

On physical examination, he was a rather well nourished man with tenderness in the epigastrium and a succussion sound on compressing the stomach. The stomach on percussion showed moderate enlargement. The radiological report by Dr. Breimer was as follows:

*Presented before the Clinical Meeting of the New York Chapter of the National Gastroenterological Association, New York, N. Y., 9 April 1951.

"The stomach is a little larger than average in size with a moderate amount of retained secretions evident before the patient had been given the barium meal.

"No structural abnormalities could be found referable to the stomach. There was marked pylorospasm, and although gastric peristalsis was very vigorous, the barium left the stomach intermittently and in very small amounts.

"The duodenal cap is very spastic and deformed, there is a small bud-like projection, containing air and barium, measuring a little less than one cm. in diameter, on the superior aspect of the cap. This I believe to be either an ulcer crater or a small walled-off perforation.

"The six hour examination showed about 30 per cent gastric retention with the remainder of the meal distributed throughout the small intestines and the proximal colon.

"Twenty-four hour examination showed the stomach distended with the patient's breakfast and mixed with barium retained from the meal the day before. There are probable occasional diverticula along the descending colon."

Dr. Breimer's conclusion was, "Active duodenal ulcer with six-hour retention."

On April 26, 1949, I performed a subtotal gastrectomy on this patient at the Fifth Avenue Hospital.

An upper transverse abdominal incision was made, transecting the right rectus muscle and retracting the left. On opening the peritoneum, a good sized inflammatory mass was seen in the region of the duodenum, which on examination proved to be a small abscess. The stomach was transected between clamps high on the left side, then dissected downward. It was difficult to dissect the duodenum from the pancreas and it was obvious that the ulcer had penetrated upwards. The stomach was removed in two pieces as the lower segment of duodenum could not be dissected free until the ulcer had been dissected off the pancreas. The duodenal stump was closed with two layers of interrupted black silk inverting sutures. The omentum was dissected from the transverse colon. Because the jejunal mesentery was very short it was decided to do a retrocolic anastomosis. The mesocolon was penetrated and a short loop gastrojejunostomy performed with the proximal portion of the jejunum toward the lesser curvature; Hofmeister modification using Von Petz clamp. The anastomosis was done in three layers anteriorly and three layers posteriorly. The anastomosis was then drawn down through the mesocolon and the mesocolon was sutured to the stomach about 1 cm. proximal to the anastomosis. The stoma readily admitted two fingers. Through a stab wound in the right flank, two cigarette drains were inserted to the duodenal stump. The abdomen was closed in layers, using continuous chromic for the peritoneum and transversalis fascia, interrupted silk for the rectus muscle, rectus sheath, deep fat and skin.

The patient had an uneventful recovery and on May 29th, the follow-up note was as follows:

"Patient feels fine. Digestion is perfect. He has practiced deep breathing since operation. He has gained five pounds. The only complaint is that he has some weakness in his legs. On examination the wound is firm and there is no tenderness."

About June 15th, however the patient began to have epigastric pain and became jaundiced. He had profuse itching. He was getting Vitamin B_{12} and liver injections. His appetite was good but his food tasted unappetizing. His weight loss was 8 to 10 lbs. since going on a low-fat diet. The bowels were regular and brown in color. The urine remained dark and was getting darker during the past week.

Diagnosis:—Serum hepatitis.

He was admitted to the Doctors Hospital and placed on a Sippy diet. The fever subsided a day after admission and his jaundice gradually receded. After 11 days he was discharged with normal appearing stools and with very little jaundice. When he arrived home he had another chill and fever and he refused to return to any hospital.

From that time until July 21, 1949, he had recurring attacks of chills and fever with increasing jaundice until he was started on aureomycin. After that, he had no fever but the jaundice remained unchanged.

On July 29th, because of the recurring jaundice and because of the epigastric pain, I operated upon him again at the New York Hospital.

An elliptical incision was made, excising the previous scar. On entering the peritoneal cavity, very dense adhesions were encountered and it was hard to get landmarks. The omentum was fatty and was excised wherever it interfered with exposure. Finally the common duct was isolated. It was dilated and stay sutures were inserted. The common duct was entered but it was impossible except at one time to pass a dilator through into the duodenal stump. There were also so many dense adhesions around the duodenal stump that its isolation was difficult. The patient lost a good deal of blood and it was felt that very possibly, as one dilator had passed through the papilla of Vater, if the duct were drained for a considerable period of time, the inflammatory process might subside, allowing free entrance of bile into the duodenal stump. One T-tube was sutured into the common duct and two cigarette drains were inserted. The peritoneum was closed with continuous chromic. The remainder of the wound was closed with through-and-through silver wire sutures and interrupted silk.

A cholangiogram was taken after the T-tube was inserted and did not show any bile entering the duodenal cap.

At the time of operation, the patient had lost a lot of blood and it was felt that with a continuous common duct drainage, his stricture might subside without further procedure. Unfortunately his T-tube was inadvertently pulled out ten days after operation. He was discharged from the New York Hospital on August 14th, but on August 23rd, he was readmitted and put on a gastric diet which he tolerated fairly well. However, he complained of severe, stabbing L.U.Q. pain following a meal on the evening of August 24th and physical examination disclosed a 5 x 5 cm. area of acute tenderness and spasm and rebound tenderness in the left upper

quadrant just below the costal margin. The remainder of the abdomen was soft and no referred rebound tenderness was found.

The patient was placed on penicillin, streptomycin, supplementary infusion and nothing by mouth. He improved rapidly and again was started on a diet of 30 c.c. of milk and cream; amphojel and gastric lavage twice a day; phenobarbital and atropine. Gradually the biliary drainage decreased and the wound healed without further treatment. An x-ray report from Dr. Weintraub revealed that the patient had a jejunal ulcer and also small diverticula in the jejunum in the immediate proximity of his gastrojejunostomy. It was felt that he had a near perforation. He was placed on a Sippy diet after having had nothing by mouth for a week. He improved gradually. His urine became brighter. He lost his epigastric pain and his jaundice diminished.

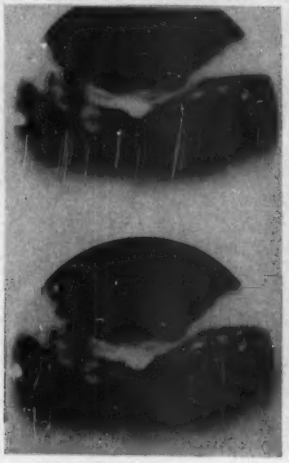


Fig. 2—Penetrating ulcer, probably in stoma, observed on second hospital admission.

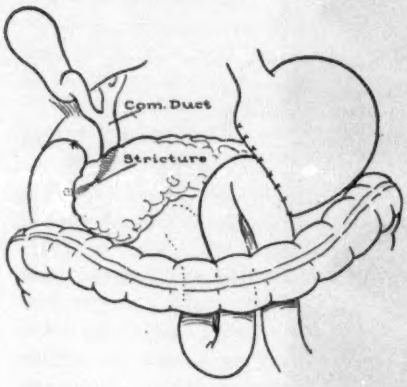
On September 24th, the patient was discharged home because his fistula had stopped draining, there was no jaundice, the stools and urine were normal and he was getting along fairly well on an advanced Sippy diet. On October 21st, 1949, however, his follow-up notes were as follows:

"Fluoroscopy and G.I. series done by Dr. Weintraub on October 20th did not disclose the jejunal ulcer seen on previous examinations. However, Mr. T. continued to have bouts of fever and intermittent discharge from the abdominal wall sinus tract. There has been no increased Jaundice; in fact, the icterus index is lower than it has been at any time since his jaundice a few months ago. His wife reports that his wound has drained for two days and she thinks it is bile, but his stools are brown. He has no elevation of temperature and he feels fairly well."

Because of repeated attacks of jaundice, chills, vomiting and loss of weight, the patient was readmitted to the Doctors Hospital on November 17th and the following operation was performed:

Exploration of common duct, anastomosis of duct to duodenal stump.

An oblique upper rectus incision was made excising the previous scar. There were extensive adhesions encountered but finally the common duct was isolated and it was possible to mobilize the duodenal stump. The common duct was transected just above the pancreas. A tube was passed up into it and down into the duodenal stump and out again at a lower portion of the duodenum, as in a Vitzel gastrostomy. The tube was brought out through the lateral margin of the incision. It had a lateral perforation in it so that bile coming down through the common duct might enter the duodenum. End-to-end suture with interrupted silk was made between the end of the common duct over the tube and the duodenum. The posterior closure line was not as good as one would wish but anteriorly and later-



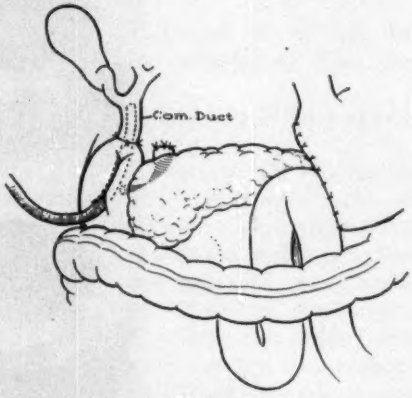


Fig. 3—Left. Artist's sketch showing local pathology.
Fig. 4—Right. Artist's sketch of operative procedure.

ally closure was effected with interrupted silk sutures. Two cigarette drains were placed in Morrison's space and drawn out with the tube. The tube was sutured into the skin and the wound closed with continuous chromic in the peritoneum, figure-of-eight through-and-through silk sutures and fine silk sutures for the rectus fascia.

During the operation the gallbladder was inspected. It was surrounded by adhesions but did not look abnormal and was not removed.

Following the operation the patient discharged bile, not only through his tube in the common duct but around the cigarette drains. These were removed and a sump drain inserted with aspiration. At the end of ten days, the drainage had ceased around his T-tube and an x-ray was taken which was described by Dr. Tillinghast as follows:

"Cholangiogram, December 1st, 1949:

"Examination after instillation of contract material (Diodrast) through a tube leading into the duodenum and common duct shows filling of the common duct and hepatic radicals.

"Fluoroscopically, the contrast material entered these structures promptly, but none of the contrast material is visualized in the duodenum.

"Delayed films with the tube clamped off, show partial emptying of the hepatic radicals and common duct, but there is still no dye in the duodenum.

"Contrast material appears to pass down along the course of the tube and outside of the duodenum at the site of entrance of the tube. It would also appear that the opening in the tube is now outside of the lumen of the duodenum.

"The contrast material is dispersed through the adjacent soft tissues in the region of the course of the tube with no distinct localized collection.

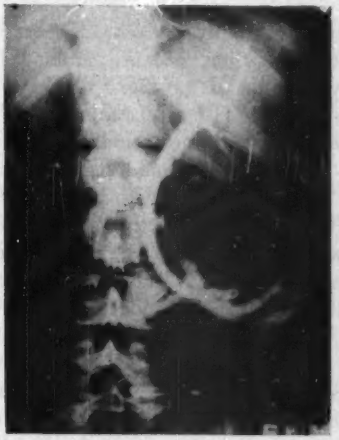


Fig. 5—Cholangiogram before removing tube in common duct.

"The tip of the tube is well up in the common duct near the bifurcation."

"*Impression:*—Hepatic radicals and common duct well visualized. No contrast material in duodenum."

Ten days later, the tube in the patient's common duct was withdrawn. He drained bile for about two days, then his sinus gradually closed.

Since that time, he has gained 40 lbs. in weight, has not had any epigastric pain, no recurrence of jaundice nor any indigestion, and his general condition is excellent.

Follow-up note taken on September 20, 1950, showed that the patient's weight was 182. He is back at work full time. He feels well. His bowels move every day. He has good color and his appetite and digestion are excellent.

This case is reported because it shows the complications that may ensue in a duodenal ulcer case and I report it with some hesitation as I realize the errors

in diagnosis and the length of time that was lost by the patient before an adequate, final operation was performed which corrected his defect.

Summary

This patient presented throughout the course of his illness a number of baffling conditions.

1. His jaundice occurred six weeks after the original gastric resection and therefore could not have been due to injury to his common duct at the time of operation but was probably caused by a secondary inflammatory reaction in the region of his duodenal stump.

2. He presented evidence on his third admission to the hospital of a penetrating jejunal ulcer which almost necessitated operation. After careful medical care his ulcer symptoms disappeared and late radiological evidence did not show a niche.

3. He might have been cured by his second operation if a nurse had not inadvertently pulled out his T-tube, because prolonged drainage might have allowed his inflammatory reaction to subside.

4. He was always a problem. His fistula would close; he would have brown stools, jaundice would diminish and one would feel that perhaps he was being cured, when again chills and jaundice would recur and the fistula would break open and discharge bile.

5. He was eventually cured by transecting his common duct and implanting it in the duodenal stump as described above. The tube was inserted into his common duct and attached so that it would be withdrawn eventually because it was felt that if it were left with the cut off in the duodenal stump, without the passage of food to help it along, it might become encrusted with bile and again cause an obstruction.

A great deal of credit must be given to the fortitude of the patient who took everything on the chin and fought from start to finish.

PRIMARY ULCER OF THE JEJUNUM

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and
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Primary or simple nonspecific ulcer of the jejunum is a relatively rare condition. Although the jejunal ulcer which develops subsequent to a gastrojejunal anastomosis is not uncommon, ulceration of the jejunum in the absence of such previous surgery is indeed unusual. The purpose of this paper is to review the literature on the subject and to report an additional case of nonspecific jejunal ulcer with melena complicated by an acute myocardial infarction.

Combes[1], in a thesis at Toulouse in 1897, first reviewed the subject of nonspecific ulcer of the small intestine. He stated that Matthew-Baillie described the condition first in 1805 and that Cruveilhier first applied the term simple ulcer. Combes collected and described thirty-six cases. In seven of these the ulcers were located in the jejunum, and the ileum was involved in twelve cases. In 1921 Judd[2] made the statement that not a single instance of primary jejunal ulcer had been observed at the Mayo Clinic up to that time. In 1922 Richardson[3] recorded the first case in this country, and he described an additional one taken from the records of the Massachusetts General Hospital. Nineteen cases of ulcer of the small intestine were reported by Oudard and Jean[4] in 1925.

Ebeling[5], in a comprehensive review of the literature, found only forty-six cases of primary ulcer of the jejunum reported from 1827 to 1932. Ebeling's case was the first in which the diagnosis of actual jejunal ulceration was made prior to operation and merits further discussion. Roentgenological study showed stasis in the duodenum and jejunum with obstruction in the first part of the jejunum. Jejunal ulcer was the preoperative diagnosis. Operation revealed a stenosing lesion involving almost the entire circumference of the bowel and causing a partial obstruction six inches from the ligament of Treitz. Pathologic examination showed an annular ulcerating lesion. The ulcer was shallow, and its base was covered with small blood clots. On microscopic sections the ulceration was seen down to the muscularis mucosa.

Buckstein[6], in 1932, reviewed a number of cases in the literature and described one of his own in which the diagnosis was made by radiographic study four days prior to the development of clinical evidence of an acute abdomen. At operation a markedly narrowed area with proximal dilatation and distal contraction was observed in the midjejunum. A small perforation on the mesenteric border of the jejunum was also evident. Buckstein advised a fractional method of studying the small intestine in the roentgen diagnosis of organic lesions of the small bowel.

In the years from 1932 to 1940 twelve cases[5-10], were reported in the literature. Berry and Dailey[7] stated that their case reported in 1940 brought the total

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number in the literature up to 76, covering a period of more than 100 years. The patient described by Berry and Dailey was similar in some respects to our own. There was evidence of melena preceding an attack of severe acute abdominal pain. The lesion was considered to be penetrating because of the associated acute pain. At surgery, blood in the intestines could be seen as a purplish discoloration through the transclucent wall of the ileum. This ended rather abruptly in the lower portion of the jejunum. Six or seven centimeters beyond this point an indurated area of irregular outline could be palpated. On opening the jejunum, an annular defect of the mucosa was found about eighteen centimeters below the ligament of Treitz measuring about 1 by 6 centimeters and bleeding actively.

During the last decade only one case of primary jejunal ulcer, that by Dowdle[11], can be found in a survey of the literature. Dowdle reported the occurrence of chronic dilatation of the duodenum produced by obstructive multiple jejunal ulcers. Therefore, it would appear that our case brings the total number reported to 78.

Case Report

C.S., a white female, age 85 years, was admitted to the hospital complaining of severe substernal pressure with radiation down both arms of approximately six hours' duration. For a period of three to four days prior to admission she had passed dark red blood by rectum in practically each stool. Two days before admission she complained of midabdominal discomfort which increased somewhat in intensity. There was associated nausea and vomiting for 24-36 hours previous to admission. Upper abdominal and epigastric distress one to two hours following meals with bloating, belching, gaseous distention, heartburn, and a "congestion" in the lower sternal region had been present intermittently for seven to eight years. Anginal pains had occurred occasionally since "a heart attack" approximately three years ago. The patient had been taking digitalis for the past year. The past history and systems review were noncontributory.

Examination revealed an agitated, restless, undernourished white female, complaining of substernal and midabdominal pain. T. 102° (R), P. 120, with frequent extrasystoles. Blood pressure 76/58. R. 32 and Kussmaul type. A cool moist perspiration was present over the skin of the forehead and extremities. Cyanosis of lips and nail beds was evident. The fundi showed mild arteriosclerotic changes. The mouth was edentulous. Eyes, ears, nose, and throat were negative except for pallor of the mucous membranes. Trachea was in midline. The chest was markedly emphysematous, and the percussion note was hyperresonant. No rales were heard. The heart was moderately enlarged to the left. The rate was rapid, and frequent extrasystoles were heard. A grade III systolic murmur was present at the apex, but no thrill was palpable. The murmur was heard with less intensity over the entire precordium. The abdomen appeared protuberant. There was tenderness to deep palpation over the umbilical region and in both lower quadrants, with questionable slight muscle rigidity. Peristalsis was active. No masses were palpable. Rectal examination revealed dark red blood on the examining glove. There was one plus pedal edema.

Laboratory data on October 7, 1950 revealed hemoglobin 9 grams (56 per cent) R.B.C. 2,700,000, W.B.C. 26,300, 3 per cent Stabs, 87 per cent Polys., 9 per cent Lymphs., and 1 per cent Baso. Urine—Sp. Gr. 1.015, Albumin and sugar negative, 2-10 WBC/hpf and few epithelial cells. N.P.N. 48.4 mg. per cent.

Electrocardiogram:—Rhythm regular. Sinus tachycardia with occasional ventricular premature contractions. Rate: (Auricular, Ventricular) 115. PR interval: 0.18. P waves upright in standard leads. QRS: 0.09. QS present in lead III: small Q present in V-6 and leads I, II. QRS complexes split in lead II; slurred in AvR, AvF. T waves: inverted in leads I, AvL; diphasic in AvF and V-3 and V-4. ST segments: sagging in lead I; elevated in V-2, V-3, V-4 and depressed in V-5. Interpretation: 1) Sinus tachycardia with occasional ventricular premature contractions. 2) Left axis deviation which is probably associated with hypertrophy of left ventricle. 3) The character of the T waves in lead I, together with the diphasic appearance of the T waves in V-4 and the negative T waves in V-5, V-6,

Fig. 1

together with elevation of the ST segments in leads V-2 through V-4, is probably associated with acute anoxia of the anterior surface of the heart, which may be due to the rapid heart rate. Serial electrocardiograms suggested.

Clinical course:—The patient continued to complain of midabdominal discomfort. A surgical consultation was obtained, and it was the surgeon's opinion that the picture did not represent a surgical condition of the abdomen. The patient was given 1,000 c.c. of whole blood, oxygen by tent, and sedation as needed. In spite of supportive therapy, the picture of moderate shock continued without apparent improvement, and the patient expired approximately eighteen hours following admission.

Postmortem Findings

General:—An approximately 115 lb. 5′ 3″, 85 year old white female is well developed and has moderate nutrition.

Peritoneal Cavity:—The surfaces are smooth and glistening and moderately to severely congested. There is approximately 400 c.c. of free serosanguinous fluid.

The intestines are moderately distended with gas. The omentum and introabdominal fat are natural. The position and relations of the abdominal viscera are natural. There are no adhesions. The intraabdominal and mesenteric lymph nodes are not apparent. All of the tissues, the aorta, the serosal surfaces of the pleura and peritoneum appear to be stained a light red, apparently due to early postmortem changes. The diaphragm is at a proper height.

Pleural Cavity:—Both pleural cavities are normal in size and contain a slight amount of serosanguinous fluid and a few firm adhesions.

Lungs:—The right lung weighs 450 grams, is normal in size and has a well aerated consistence. The bronchi, blood vessels and lymph nodes are natural. The lower lobes have a moderate degree of congestion. The left lung weighs 400 grams and is essentially similar to the right.

Heart and Blood Vessels:—The heart is moderately increased in size with hypertrophy of the right and left ventricles. The heart weighs 600 grams. The aortic valve leaflets are thickened and somewhat calcified but appear relatively competent. The mitral valve leaflets are slightly thickened. The remainder are natural. The endocardium is natural. The left coronary artery at its proximal ¼ is considerably atherosclerotic with a small patent lumen. The right is natural for the age. The ostia of the coronary arteries are natural. The valve rings are natural. The ventricular walls are moderately increased in thickness anteriorly. In the region of the apex the left ventricle has numerous tiny hemorrhagic areas alternating with whitish zones. The aorta is moderately atherosclerotic. The pulmonary artery, superior and inferior vena cava are natural.

Gastrointestinal Tract:—The esophagus and stomach are normal. Approximately 15 cm. distal to the ligament of Treitz there is a 1.3 cm. sized punched-out ulcer which goes down to serosa. On the serosal side it is covered with some fibrinous exudate. Approximately 5 cm. of surrounding adjacent jejunum is thickened, and the mucosa in this area is grayish-brown in color and granular to the touch. The mucosal folds are absent in this area. The entire distal portions of the small and large intestines contain variably changed blood. Near the distal end of the jejunum is a diverticulum which measures approximately 2 cm. at the base and extends approximately 5 cm. outwardly as a pouch. The wall here is thin.

All other organs are essentially natural for the age.

PATHOLOGICAL DIAGNOSES (GROSS)

Cardiovascular System:—

1. Atherosclerosis of the coronary arteries, particularly the left, moderate, old.
2. Atherosclerosis of the aorta, moderate, old.
3. Myocardial infarction, anterior, moderate, recent.

Respiratory System:—

1. Bilateral pulmonary congestion, light to moderate, recent.

Gastrointestinal Tract:—

1. Acute primary penetrating jejunal ulcer, severe, recent.

2. Gastrointestinal hemorrhage, severe, recent.
3. Jejunal diverticulum.

Microscopic Description

Jejunum:—The mucosa in one area is completely denuded and there is a punched out crater. The lining is necrotic fibrin and granulation tissue. A moderate infiltration with mixed inflammatory cells extends through the wall. A few follicular collections of lymphocytes are present. The submucosa is slightly fibrosed, and the subserosa is edematous. There is no evidence of malignancy. A similar but lesser inflammatory process extends into the adjacent jejunum for a distance of about 5 cm.

Myocardium:—There are numerous areas of necrosis of muscle fibers with many infiltrating polys.

Lungs:—Many of the alveoli are filled with clear fluid.

Microscopic diagnoses are the same as the gross.

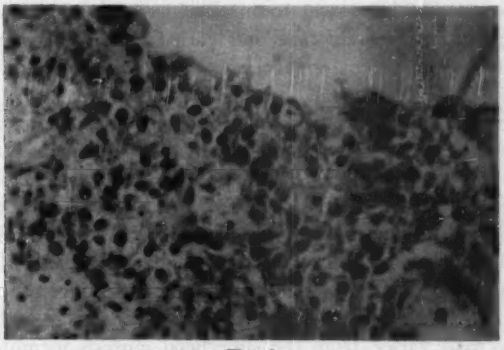

Fig. 2

Etiology and Pathogenesis

The etiology of nonspecific ulcer of the jejunum is as obscure as is that of the much more common gastric or duodenal types. Various theories have been advanced as to the cause of this lesion. Many authors mention its resemblance to peptic ulcer and suggest the same theories of origin. Heterotopic gastric mucosa has been mentioned as a possibility, but this has never been demonstrated. Gale[12] and Brown[13] favor the factor of focal infection. Brown cites the work of Rosenow on the selective affinity of certain strains of streptococci for the intestinal tract. Stitch and Collier[14] advocate the theory of vascular obstruction. Histological examination in their case showed evidence of endarteritis and thrombosis in some of the vessels of the subperitoneal coat. Bigger[15] and Taylor[16] believe that trauma may play a part in this condition. Friedman[17] advocates the role of excess or deficiency of endocrine secretion. This may act directly on the walls of vessels of the intestinal tract with subsequent devitalization through interference with their

blood supply. Ulceration may then ensue through bacterial invasion. Whatever the initial cause of the bowel necrosis in the usual ulcer of the stomach or duodenum, the acid gastric juice, although not the exciting cause, is surely a very definite contributory factor in maintaining its chronicity. In the jejunal or marginal ulcers which follow gastrojejunostomy, this factor is likewise present. After passage of gastric contents from the stomach, the hydrochloric acid is neutralized and the pepsin inactivated by the time the chyme reaches the jejunum. Therefore, it is highly improbable that this acidity is a factor in producing the jejunal ulceration. However, it is conceded that proteolytic digestion may take place through the action of trypsin and prevent repair of the ulcer by cicatrization.

Pathology

From Ebeling's[5] analysis the majority of the ulcers were located in the upper jejunum opposite to the mesenteric attachment. In most, single ulcers of the jejunum were present, but in some they were multiple. A few showed associated ulcers of the ileum, and occasionally there were associated gastric and duodenal ulcers. The lesion occurs in greater frequency in the male with a ratio of about three to one. Age in the reported cases has varied from eight years to eighty-five years in our own patient, the oldest age previously recorded being seventy-seven. The majority occurred between the ages of forty and sixty.

The descriptions of the ulcers were similar to those of chronic peptic ulcers occurring in the duodenum or stomach. They were clean-cut and punched out. In cross-section a terrace formation of the edges was seen. The size varied from a few millimeters to two to three centimeters. The degree of induration was variable. The perforated ulcers usually presented only a minute opening. The configuration was mainly circular and occasionally annular. Stenosis of varying degree of bowel with induration was commonly present at the site of the ulceration.

Microscopically, the ulcers showed a variable degree of round-cell infiltration and fibrosis. Necrosis varied from the superficial mucous membrane destruction to involvement of the submucosa and deeper layers, and to actual perforation through the serosa. Few writers have recorded the presence or absence of enlarged regional nodes. In those instances in which the glands were examined microscopically, the changes noted were those due to chronic inflammation.

Symptomatology

Symptoms vary widely, but many of these cases resembled closely those of gastric and duodenal ulcer. In some patients there was pain one to three hours after meals and during the night, the pain being relieved by alkali. There were all gradations of dyspeptic symptoms without the classical periodicity and rhythmicity of gastric or duodenal ulcer. Some complain only of indigestion or "stomach trouble" for months or years. Hematemesis occurred in two recorded cases (Schmilinski[18] and Murphy[19]). Melena was seen in two other instances besides our own. Very few of the reported cases sought relief until obstructive symptoms or perforation occurred. In those with perforation, the location of the pain is at first in the midabdomen but rapidly becomes generalized. Board-like rigidity

rapidly appears similar to that seen in perforated peptic ulcer. The great majority of cases terminated in spontaneous perforation. In those ulcers which caused stenosis, subacute obstructive symptoms were present, as cramp-like pain, constipation, belching, distention and vomiting with some relief by lavage.

Diagnosis

Clinically, the presence of simple jejunal ulcer is difficult to detect. Of the reported cases, only two have been diagnosed preoperatively (Ebeling[5] and Ravdin[20]). A clear and concise clinical picture is usually not available. Gastrointestinal studies would appear to be extremely valuable. Upon roentgenological examination, regurgitation of the opaque medium into the bowel proximal to the ulcer, actual distention of the proximal loops in the presence of obstruction, and demonstration of a narrow constricted lumen at the site of stenosis might be expected.

Buckstein advocates special studies of the small intestine, according to the following procedure: after ingestion of the barium, several films of the stomach and duodenum are taken; a 14 x 17 film is then taken every 15 minutes during the period of one hour. This is followed by a similar examination at two, three, five, six and nine hours. One author has stated that chronic duodenal ileus should lead one to think of jejunal ulcer. This diagnosis can be made only by roentgenological studies.

In the diagnosis of perforation, determination of air under the diaphragm would be extremely valuable. Although the jejunum is retro-omental, air, with the patient in the upright position, will find its way to the dome of the diaphragm.

Treatment

Unlike peptic ulcer there is no so-called medical treatment, inasmuch as the possibilities of medical treatment in the chronic ulcer manifested by pain or obstruction are practically nil. It would appear, from autopsy reports, that some people may go many years without being aware of this disease. When the diagnosis is made, there is no alternative to surgical treatment. Clinical signs of perforation demand, of course, that immediate surgery be undertaken. Since so many of the reported cases have gone on to perforation, resection of the jejunum offers the best prognosis. The question of malignancy must be kept in mind in cases where an annular growth with enlarged mesenteric glands is found.

Summary

1. Simple or nonspecific ulcer of the jejunum is a rare condition found fortuitously.

2. A pertinent review of the literature of this subject is presented.

3. A case of nonspecific ulcer of the jejunum with melena complicated by an acute myocardial infarction is described.

4. The etiology, pathology, symptomatology, diagnosis, and treatment of this lesion is discussed.

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RESULTS OF TREATMENT OF BLEEDING PEPTIC ULCER*

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The treatment of bleeding peptic ulcer has for years been the subject of controversy. Figures have been presented as by Meulengracht[1] and adherents[2] to show a minimal mortality under a straight medical regime while others, as Finsterer[3] and in this country Stewart[4], recommended almost routine early surgery and give partial gastrectomy as the treatment of choice in major gastric hemorrhage. The literature is replete with courses recommended between these extremes so that the surgeon, faced with an occasional massive gastric hemorrhage, is almost as terrified as the patient when he realizes that he does not have a solid plan of procedure.

It is not our purpose to review the literature nor to give or evaluate the varied advices of others[5]. It is planned here to present briefly the results at the Colorado General Hospital over the last twelve year period and to add a few of our personal impressions. These patients were under the care of many different staff men and no uniform course of treatment had been adopted.

There were 86 cases (Table I), admitted for bleeding peptic ulcer, most of them directly to the medical wards. Ages ran from 21 to 84, with an average of 54.4 years, which I believe is a higher average than in most reports. The sex and location are about in line with the average.

TABLE I
86 Consecutive Cases Of Bleeding Peptic Ulcer Admitted To The Colorado General Hospital From 1939 Through 1950

Ages	21 to 84 years Average 54.4 years	
Sex	Males	70
	Females	16
Location	Duodenal	67
	Gastric	11
	Stomal	4
	Undetermined	4

It is most significant that the average duration of ulcer symptoms was over 13 years (Table II) though an occasional patient gave a very short history. It is also most significant that the average duration of bleeding before admission was 3.7 days—which precludes prompt treatment in the average case.

As in all reports, the mortality varied with age (Table III). The total mortality of 19.7 per cent is high and may be partially explained by the large number of patients over 60. But, in the individual case, age cannot influence the final decision as to treatment. One patient of 26 died following surgery while on the other hand there were medical and surgical recoveries in patients over 70.

Most criteria of amount of blood loss are inaccurate. Determination of the red cell circulating mass has not been available routinely here. From other reports[6,7], this determination would seem to be the only truly accurate measure

*Read before the Sectional Meeting of the American College of Surgeons, Denver, Colo., 7 April, 1951.

of blood loss. Drop in blood pressure and fainting, especially if repeated, suggest severe blood loss. But neither blood pressure nor red cell count can be considered more than a rough guide to estimation of blood loss as vasoconstrictive shunts and delay in hemodilution may keep the pressure and count somewhere near a normal range during the earlier periods of severe hemorrhage. It will be seen here (Table IV) that the fatal cases had a considerably lower average red cell count on admission than did the recoveries and yet the range is wide. The individual count may be based more on duration than on amount of blood loss and it gives no clue as to arrest of hemorrhage.

TABLE II

DURATION BEFORE ADMISSION

Ulcer Symptoms	
Maximum	40 years
Minimum	3 days
Average	13.4 years
Bleeding	
Maximum	14.0 days
Minimum	1 hour
Average	3.7 days

The medical mortality (Table V) of 21.2 per cent and the surgical mortality in a lesser amount of 15 per cent are subject to so many variables that the figures prove very little. The medical staff feel that many of their cases were received late and in an irreversible stage and so raised their mortality rate to a level that does not give a fair report of medical treatment. The surgical staff in turn consider that their cases were the late ones that had failed under medical management and so represented the poorer risks.

TABLE III

MORTALITY AS TO AGE GROUPS

Ages 20 to 40	24 cases	2 deaths — 4.8%
40 to 60	41 cases	8 deaths — 19.5%
Over 60	21 cases	7 deaths — 33.3%
TOTAL	86 cases	17 deaths — 19.7%

The nonoperative deaths numbered 14 (Table VI). Two were admitted in extremis and treatment was barely started. In another three, the true diagnosis was concealed by other conditions and not made before autopsy. The other nine were treated under a regime that included blood transfusion, diets, antacids, atropine and sedatives. No attempt is made here to evaluate these measures. On study of the charts of these nine cases, it appears in retrospect that six were probably operable at some time. That is a personal opinion and in no way proves that results would have been different.

TABLE IV

MORTALITY AS TO ADMISSION RED COUNT

Recoveries:		
	Minimum	1.7 million
	Maximum	6.1 million
	Average	3.6 million
Deaths:		
	Minimum	1.5 million
	Maximum	4.2 million
	Average	2.6 million

An average of 54 years (Table VII) is not too favorable for surgical treatment of severe hemorrhage and probably hurries the operative procedure. While gastric resection is admittedly the treatment of choice from a purely technical angle, it must be considered that the local excisions, though in some more radical surgery was required later, were probably life-saving. Vagotomy cannot be trusted for positive arrest of hemorrhage.

Of particular interest should be the analysis of the three surgical deaths (Table VIII). Gastroenterostomy has been notorious for its failure to stop hemorrhage. Bleeding did not stop in the two cases so treated which probably means that a reduction in acid does not suffice to allow healing of a large eroded artery. The

TABLE V
MORTALITY AS TO TREATMENT

Cases Treated Medically	66
Deaths	14
Mortality	21.2%
Cases Treated Surgically	20
Deaths	3
Mortality	15%

other death emphasizes more the danger of dicumarol than that the gastric resection was inadequate.

A cool analysis of these figures does not solve the question of how to treat bleeding peptic ulcer though it may show the cause of some failures. In retrospect, opportunities for success may often be seen which were not always apparent at a given moment.

DISCUSSION

Early diagnosis is essential. Not all cases are seen early. Accurate early diagnosis is not always possible when surgery would do the most good. Ulcer craters in stomachs not empty of blood cannot uniformly be shown even if early x-ray is

TABLE VI
ANALYSIS OF 14 MEDICAL DEATHS

2—admitted moribund
3—presence of hemorrhage not recognized, admitted for other conditions
9—treated expectantly—received from 0 to 10,000 c.c. blood
Of these 9—
3 never operable
6 probably operable at some time.

considered justifiable. The very great majority of these cases gave an ulcer history. In those that do not, and in which the diagnosis is sufficiently in doubt, it still seems that conservative treatment offers the best prognosis regardless of recent advice[1,2] to the contrary.

Transfusion remains the key to success in this emergency. First, it is hard to find a better rule than that of arranging immediate blood transfusion for the bleeding ulcer patient, then to give 500 c.c. of blood every 6 to 8 hours to stabilize him. If he won't stay stabilized by the end of 24 hours as judged by appearance, blood-pressure or repeated hematemesis, he becomes a surgical candidate with blood going before, during and after surgery. Second, if a patient stabilized at the end of 24 hours again starts to bleed, he should be considered a surgical candidate

as soon as he can be made ready. Third, if he is stabilized in 24 hours (and that figure is not too exact as he may have been bleeding for days before admission), then he is entitled to medical treatment, to a build-up and x-ray before making a decision as to surgery. Fourth, in those patients admitted for a second or third hemorrhage, who become stabilized, the risk of surgery would seem far less than that of still another hemorrhage. On the other hand, universal surgery in all severe

TABLE VII

ANALYSIS OF SURGICAL RECOVERIES

17 Cases	Average age 54.2 yrs. No operation within 48 hours of admission.
Operations	1 Vagotomy 5 Local excisions or closures. 2 required resection later 11 Partial gastric resections

peptic ulcer bleeding, it seems fair to state, will result in a much higher mortality than selection along this general line.

If surgery is done, the main rule is, as shown here, to control the bleeding point. Gastric resection is good because it excises the bleeding ulcer and usually obviates future recurrence. Gastric resection without ulcer removal is not good though in some low duodenal ulcers, tamponade must be submitted. Gastroenter-

TABLE VIII

ANALYSIS OF 3 SURGICAL DEATHS

Case 1	Age 39	Bleeding 7 days	PGE	Continued bleeding.
Case 2	Age 71	Bleeding 1 day	PGE	Continued bleeding Peritonitis
Case 3	Age 50	Bleeding 2 days	G R	Recurrent bleeding after dicumarol. Stomal ulcer found at second operation. Died of hemorrhage.

Average age 53.

ostomy without ulcer removal is worse. Ligation of vessels leading to an ulcer is of no added value. In extreme cases or under unsuitable conditions, it is still good practice to remove an ulcer locally and wait for a better day to do the definitive surgery. Under all these conditions, adequate blood transfusions not only to keep up circulatory volume but also to prevent hypoxia may be the difference between success and failure.

CONCLUSIONS

1. It is appreciated that the mortality figures for the series discussed here are much higher than the average report. The figures may be more representative of the average clinic hospital than many of the good results reported. Lateness of admission or inadequate treatment are the two factors to be considered.

2. The cause of failures are presented and may give lessons for future success.

3. Indications for operative or conservative treatment are presented and a rational standard treatment suggested that should greatly improve these results.

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PERFORATIONS OF THE SMALL INTESTINE DUE TO NONPENETRATING TRAUMA

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Traumatic perforations and rents in the small intestine occur when an unexpected blow strikes the abdominal wall. When a blow is received unexpectedly the muscles of the abdominal wall are not contracted. This lack of muscle "splinting" leaves the viscera unprotected and exposed to the full power of an assailing line of force. Therefore the abdominal viscera have little protection against injury under these circumstances. Usually the subcutaneous tissue and the abdominal muscles absorb the shock of blunt trauma, thus diffusing and lessening the power of a blow.

When the small intestine becomes the victim of blunt trauma, there are several areas most often involved. These are the fixed segments:

1. The duodenum
2. Near the ligament of Treitz
3. Near the ileocecal region.

When blows to the epigastrum injure the duodenum, the third portion is most frequently affected. This occurs because it is attached to the bodies of the upper lumbar vertebrae by peritoneum. A sudden striking force directed toward the epigastrum will drive the posterior wall of the duodenum against the surface of the vertebral bodies producing an injury to the individual wall. Bleeding may occur in some instances. The blood has been known to escape into the retroperitoneal spaces.

The same general factors produce rents in the vicinity of the ligament of Treitz. A potent force striking a segment of jejunum near this ligament moves the loop beyond its limit of mobility, thus producing a tear. The other most frequent location for traumatic laceration is within two to three feet proximal to the ileocecal valve. These lesions result when the external force compresses the ileum against the vertebral column or the pelvic bones. Another contributing factor may be the increase in intraabdominal pressure from external violence. How much the sudden compression of fluid or gas within an intestinal loop contributes to the etiology of this injury is purely speculative.

A study of traumatic perforations of the small intestine at the hospitals with which I am associated is the basis for the present study. From January, 1934 to January, 1951, thirty authentic cases, proved by operation or at autopsy, were encountered.

An analytical study of these cases brought to light many salient factors of importance. The most important single contribution made by the patient, which aids in arriving at a diagnosis of intestinal perforation, is the history of trauma followed by abdominal pain. Either continuous or intermittent pain which is

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persistent, associated with vomiting or hematemesis, may indicate visceral injury. The examiner should not be deceived by a posttraumatic period of freedom from symptoms.

Examination of the patient in most cases suggests the presence of an acute surgical abdomen. Where a decision cannot be reached as to operability, frequent repeated physical examinations are important. The pulse and blood pressure should be recorded hourly. An accurate diagnosis may be reached occasionally by means of a flat plate x-ray of the abdomen for the presence of free air under the diaphragm.

When the diagnosis of small bowel perforation has been made surgery is imperative after an adequate preoperative preparation to combat shock, etc.

Following the surgical repair of the traumatic perforations antibiotics were used. The mortality is high in this type of injury; in our first 18 cases only eight survived (1934-1943). A contributing cause was the multiple injuries seen in this group. In the majority of our cases the perforations in the small intestine were the outstanding injury and with its complicating peritonitis was the cause of death. The lowering of this high mortality resides with the surgeon. A decision for or against operation in traumatic abdominal injuries should be made as soon as possible. Ideally, 4-6 hours after the accident is the most favorable time. Delay, lingering, and waiting are the most common reasons for a fatal outcome. The longer the delay, the more unfavorable is the prognosis. This delay may be due to the patient's failure to seek early medical care. Occasionally the delay is caused by the surgeon who fails to evaluate the serious nature of the injury.

EDITORIALS

Pyloric Spasm

It was interesting to read an article by Roka and Lajtha* regarding the administration of 100 c.c. of a 1 per cent solution of procaine orally in pyloric spasm. We have been using a 1 per cent solution of larocaine for many years, advocating the application to the nasal mucosa and the posterior pharynx before passing a duodenal tube through the nose or, when the tube was to be passed orally, the application of the solution to the back of the throat by means of a swab or spray. In patients who had a tendency to nausea, vomiting, pain, spasm or closure of the pylorus due to irritation or inflammation, the administration of 50 to 75 c.c. of a 1 per cent solution of larocaine relieved the distress. We have also used procaine the same way, and found that it was safe to repeat the medication once or twice daily without fear of habit formation. Naturally, where the obstruction was due to cicatrization and complete closure, the relief was temporary.

We are in agreement with Roka and Lajtha that oral administration of one or the other of these medicaments is a simple and safe therapeutic procedure which may be used in functional spastic pylorus and as a preparation for surgical intervention in organic obstructions of the pylorus.

*Roka, G., and Lajtha, L. G., Brit. M. J. **2**:1174, 1950.

Samuel Weiss

Belladonna Preparations as a Cause of Glaucoma

Physicians may not be aware of the danger of provoking an incipient or latent glaucoma in patients who do not manifest definite clinical symptoms.

This is especially pertinent in reference to patients between the fourth and sixth decades of life. They consult the physician for gastrointestinal complaints, such as duodenal or gastric ulcers, ulcerative or spastic colitis, hyperacidity or psychosomatic syndromes referable to the alimentary tract. If the drug is taken, ocular symptoms may follow immediately, in a few days, or even several weeks later.

Because of the possibility of these occurrences, it is admissable for the prescribing physician to question the patient as to possible past eye disease or eye treatment. We have been able to trace two patients who definitely developed glaucoma as a result of the administration of a belladonna preparation. Neither of these patients had had any inkling, or promonitory symptoms, of his eye condition.

Samuel Weiss

BOOK REVIEWS

MONOGRAPHS ON SURGERY—1950: B. Noland Carter, M.D., Ph.D., Editor. Professor of Surgery, University of Cincinnati; Director of the Surgical Services, Cincinnati General Hospital; Advisory Editors, J. V. Meigs, M.D., C. Huggins, M.D.; A. R. Shands, M.D. 501 pages. Thomas Nelson and Sons, New York, 1949. Price $12.50.

A bound volume of monographs by different authors will appear annually similar to this present volume. This "1950 Monographs on Surgery" in a bound volume represents a distinct change in the policy of the editors and publishers of Nelson's Loose-Leaf Surgery.

This initial volume contains contributions by W. A. Altemeier on "Chemotherapy in Surgery". Altemeier refers to Paster and Joubert's (1877) observation of bacterial antagonism between certain airborne bacteria and the anthrax bacillus, the growth of the latter being inhibited by that of the former. They also suggested that this antagonism could be used in the treatment of certain infections. Reference is made to lysozyme (1922), sulfanilamide penicillin from Penicillium Notatum prontosil, streptomycin from streptomyces griseus, chloromycetin from streptomyces venezuelae, aureomycin from streptomyces aureofasciens, and the various sulfa preparations and antibiotics are fully discussed, including also reference to Polymixon B(aerosperin), penatin, pyocyanine and pyocyanase, fumigacin, claviformin, dicumarol, and comments on carbuncles, osteomyelites, and empyemia, and other infections.

"Carcinoma of the Lung" by Debakey Ochsner and Decamp; "Pancreatitis" by Siler and Wulsin; "Carcinoma of the Endometrium" by L. Parsons; "Nontuberculous, Nongonorrheal Infections of the Urinary Tract" by R. Chute; and "Modern Laboratory Technics in Urology" by W. W. Scott are all well presented, and are easy to read, and to understand. Siler and Wulsin of the Cincinnati General Hospital have written an excellent monograph on "Pancreatitis". They, briefly but adequately, discuss embryology, anatomy, histology, physiology, digestive functions of the pancreas, pancreatic enzymes, control of pancreatic secretion, etc., causes of pancreatitis, cholangiography, pathology of pancreatitis, fat necrosis, calcification, symptomatology, laboratory features, differential diagnosis of pancreatitis, diagnosis, treatment, and prognosis. They include a fine list of references in the bibliography.

This initial effort by Nelson and Sons to issue bound monographs is a successful one and merits continuation.

The volume is recommended to supplement other texts on surgery and should be of interest to surgeons, orthopedists, and resident surgeons and physicians interested in the various topics presented in this volume.

INTESTINAL INTUBATION. Meyer O. Cantor, M.D., M.S., F.A.C.S., Assistant Attending Surgeon, Deaconess Hospital, Detroit, Michigan. 333 pages, 138 illustrations. Charles C. Thomas, Springfield, Illinois, 1949. Price $7.50.

Dr. Cantor's book on Intestinal Intubation is timely and well worth reading. The author describes and illustrates the development of the tube from its early inception. In addition, he gives detailed information for use in the various surgical and nonsurgical conditions. Interesting roentgen studies show why the tube fails to reach its destination, and how to overcome this obstacle.

Dr. Cantor fails to mention a duodenal tube which has been in use for more than two decades. This duodenal tube has a small piece of metal embedded into its tip. It is opaque to the roentgen rays and may be used either orally or through the nose.

This, however, should not detract from the value of this little volume, which the reviewer recommends highly.

DISEASES OF THE AORTA. DIAGNOSIS AND TREATMENT: Nathan E. Reich, M.D., Associate in Medicine, Long Island College of Medicine; Attending Cardiologist, Harbor Hospital, Brooklyn, N. Y.; Associate Attending Physician, Kings County Hospital, Brooklyn, N. Y.; Senior Cardiologist, Veterans Administration, Brooklyn, N. Y. 288 pages. The MacMillan Company, New York, 1949. Price $7.50.

This is truly an informatively and instructively interesting book on diseases of the aorta. The work was well and practically planned making it a useful text for students, general practitioners and specialists.

With modern methods of surgical treatment of coarctation of the aorta, patent ductus arteriosus, and other anomalies, and with recent advances in the use of venous catheterization, cardioangiography, and radioactive isotopes—

it is of considerable value to read and study monographs of this type. Dr. Reich has planned his monograph carefully and his effort in this direction should be recognized and appreciated. It is recommended to all physicians, cardiologists, and medical students, as a satisfactory work on diseases of the aorta.

Dr. Reich includes an interesting chapter on the history, anatomy, and embryology of the aorta. Congenital anomalies of the aorta, atherosterosis of the aorta, syphilis of the aorta, aneurysms, occlusion of the aorta, and diagnostic procedures, all receive adequate and satisfactory consideration.

The author also briefly discusses antibiotics and anticoagulants. The author erroneously states (p. 3) that "Sandifort presented the first description of the tetralogy of Fallot (1888) in 1777", Nils Stensen (1671-1672) in T. Bartholin's "Acta Medica et Philosophica" gave the first description, followed by Morgagni in his "De Sedibus" (1761). Farre, in 1814, also discussed tetralogy of Fallot.

There are a number of helpful illustrations, and a liberal number of references to the literature. Roentgencinematography, kymography, tomography, angiocardiography, abdominal aortography and fluorocardiography (electrokymography) are very briefly mentioned.

This reviewer considers this small monograph an excellent aid to a better understanding of diseases of the aorta.

TABLET MAKING: Arthur Little and K. A. Mitchell. 121 pages, 41 illustrations. The Northern Publishing Co., Ltd., Liverpool, England, 1949. Price 15/.

This little volume would be of more interest to the pharmacist than to the physician, although, the reviewer wonders whether or not the general pharmacist will find it interesting.

It is recommended that the book be introduced to pharmaceutical houses which manufacture the great volume of tablets prescribed by physicians.

A TEXTBOOK OF PHYSIOLOGY: Edited by John F. Fulton, M.D., Sterling Professor of Physiology, Yale University School of Medicine, with the collaboration of 12 associate editors and 29 contributors. 16th edition, illustrated. 1258 pages, W. B. Saunders Company, Philadelphia, Pa., 1949. Price $10.00.

This excellent and well established work has been thoroughly revised and considerably extended by new additions and greatly lengthened sections.

The chapters on the endocrines by Jane A. Russell, Ph. D., may be particularly pointed out as having been brought up-to-date. The chapters on hemodynamics, events of the cardiac circle, cardiac output, nutrition of the heart, blood pressure and pulse alone are worth the price of the book, for every cardiologist and physician interested in physiology of the circulation. The same may be said of the chapters on the physiology of digestion and secretion of the alimentary tract; stomach; small intestine; large intestine; metabolism and nutrition. These several chapters are of particular value to all gastroenterologists and students, and particularly those younger physicians and surgeons who are interested in the workings of the gastrointestinal tract. The sections on physiology of the organs of circulation of the blood and lymph; cerebrum, basal ganglia and reticular formation; cerebrum and cerebellum and the autonomic nervous system, are excellent.

The chief editor, Dr. Fulton, is to be congratulated on having selected such able contributors, and for his own fine contributions to this book.

The reviewer considers this the best available one volume textbook on physiology (in the English language) and highly recommends this textbook to all students and practitioners, to all investigators and specialists, and to all medical libraries, in hospitals and colleges.

THE ROENTGEN MANIFESTATIONS OF PANCREATIC DISEASE: Maxwell Herbert Poppel, M.D., F.A.C.R., Associate Professor of Clinical Radiology, New York University-Bellevue Medical Center, Associate Roentgenologist New York University Hospital, Associate Radiologist Mount Sinai Hospital, New York, Consultant Radiologist U. S. Naval Hospital, St. Albans, N. Y., and U. S. Veterans Hospital, Bronx, N. Y., 400 pages, fabrikoid, 166 illustrations. Chas. C. Thomas, Springfield, Ill., 1951. Price $10.50.

This liberally illustrated book on the Roentgen Manifestations of Pancreatic Disease presents a detailed roentgen approach to the detection of demonstrable pancreatic disorders. The author's extensive experience enables him to explain and interpret all the various roentgen manifestations and thus to bring their significance into sharper focus.

The appreciation and correlation of the roentgen manifestations permit a crystallization

of ideas which help to reflect the underlying basic pathological mechanisms in their various static and dynamic sequences. This often permits a pathologic translation, thereby harmonizing the diagnosis with the actual disease.

All who are concerned with the diagnosis of pancreatic disease, i.e., gastroenterologists, roentgenologists, internists, surgeons, pediatricians, geriatrists, will find this book an indispensable storehouse of roentgen information. Here, in one 400 page volume with 166 figures, is the most comprehensive report of the roentgen diagnosis of pancreatic disease ever published.

Every effort has been made to make the roentgen presentation of the pancreas as complete as the present knowledge warrants. The main guiding thought has been the presentation of the *roentgenologic survey of the anatomy, physiology and pathology of the pancreas.*

Although direct roentgen interest has been primarily focused upon the pancreas, the abnormalities (and roentgen methods for their apprehension) of adjacent and distant viscera have been carefully considered. There are special chapters on Pancreatic Infections, Primary Pancreatic Tumors, Calcareous Diseases of the Pancreas, Pancreatic Exocrine Insufficiency, Roentgen Methods, Differential Diagnosis.

The table of contents and the index are excellent. The paper, format, print and illustrations are very good. An unusual feature of the book is the setting of all the illustrations at the top of the respective pages. Many of them are of the nature of one of a kind that cannot be replaced and are considered collectors' items. In some cases they are the only existing films representative of the disease discussed.

The author tells the roentgen pancreatic story in a highly scientific style with a kind of engaging earnestness. — It is his purpose to give the reader simple roentgen and basic information as will prove useful in understanding the diagnosis of pancreatic disease.

The book presents an inclusive affirmative approach to the roentgen diagnosis of pancreatic disease. It brings integration and meaning into the complex subject.

It succeeds admirably in cutting through the outer layers of confusion towards the hard core of knowledge as regards the roentgen diagnosis of pancreatic disease.

The author gives evidence of profound scholarship coupled with wide understanding of the roentgen diagnosis of pancreatic disease.

It is undoubtedly the most unique medical text of the year—a factual book about a little known subject. It will surely find a place among the roentgen classics.

TEXTBOOK OF HUMAN PHYSIOLOGY: William F. Hamilton, Ph.D., Professor of Physiology, University of Georgia School of Medicine. Second edition. 623 pages, 139 illustrations. F. A. Davis Co., Philadelphia, Pa., 1949. Price $7.00.

This second edition of a very helpful and instructive textbook of physiology should rapidly become a most popular work for all medical students, premedical students, nurses, general practitioners, and for all those preparing for basic science board examinations, and medical boards, as well as specialty boards, and medical civil service tests. This is a compact work, containing only essential material, which every student can easily understand and remember!

The reviewer recommends this volume as an excellent compact work on physiology, by an experienced teacher of the subject, made easily readable, and quite easily absorbed!

DOOR OF HOPE: A novel. Joseph S. Diamond, M.D., Greenberg Publishers.

This is a story of the passionate struggle for liberty in a Rumanian town sixty years ago. It depicts especially the difficulties of the Jews as a people and especially the peasants against the powerful landowners. A bit of romance runs through the story which abounds in efforts and ideals for a better way of life.

The author writes in a fine vein of sentiment, humanity and philosophy. As the story carries on, writings and sayings of celebrated men of the past are quoted. Since the story is essentially one of a people crying against the crimes that culminated in the inhumanities of the Hitler period, it contains none of the subtleties of characterization and none of the complexities of technic of novels primarily literary in character. It is written first hand by a person who is acquainted with the facts and conditions existant in Europe up to recently. The "Door of Hope" should thus be judged for what it is intented to be. the life struggle of a people reaching for the light that had been denied them.

It is pleasant to have a person of Dr. Diamond's interest and ability in gastroenterological work take time out and produce something that is helpful to humankind to correct some of the errors that still exist far too abundantly in the world.

HANDBOOK OF APPLIED PSYCHOLOGY: Edited by Douglas H. Fryer, Ph.D., New York University, and Edwin R. Henry, Ph.D., Richardson, Bellows, Henry and Company, Inc., and Consultant, Standard Oil Company, New Jersey; by 116 contributors. Two volumes, 842 pages. Rinehart and Company, Inc., New York, 1950.

This monumental and exhaustive work on applied psychology in all its aspects, edited by two experts, and composed of contributions by 116 authorities, should be on the shelf for ready reading and study in all libraries, colleges and schools, and on the desk of every psychologist, sociologist, neuropsychiatrist, and teacher of psychology.

This work does provide a comprehensive survey of applied psychology, and will be a great help to all students of psychology. The subjects are well arranged, and understandingly written. The two volumes make for easier handling and reading. The printing is clear and easy on the eyes.

The reviewer recommends this work to all teachers and students of psychology, as well as to leaders in educational fields.

CLINICAL NUTRITION: Edited by Norman Jolliffe, M.D., F. F. Tisdale, M.D., and Paul R. Cannon, M.D., for the Food and Nutrition Research Council. 78 tables and 127 illustrations in full color, 925 pages. Paul B. Hoeber, Inc., New York, 1950. Price $12.00.

This splendid volume contains the work of thirty-six authoritative contributors, whose names are well-known in medical circles.

Dr. Jolliffe has devoted years to the study and treatment of nutritional disturbances. With his collaborators, he has produced an invaluable volume on diagnosis and treatment.

Stress is laid on diagnosis; there are illustrations in color, which enables the doctor to compare his patients' lesions and to institute proper therapy.

The book is divided into three main sections: Diagnosis; The Nutritional Elements; Therapy and Prevention. Additional value is added to the work by the appendix, which contains the recommended daily dietary allowances, food values, dietary patterns, weight charts, etc.

The editors, contributors, and publishers have obviously spared no pains in the preparation of this volume.

THE URINARY EXCRETION OF THE KIDNEY: A. V. Wolf, Ph.D., Associate Professor of Physiology, Albany Medical College, Union University. 363 pages. Grune and Stratton, Inc., New York, 1950. Price $7.50.

This is somewhat of an unusual volume, by an expert in this field. As a capable investigator and teacher, interested in human physiological processes, as well as pathological changes in function, Dr. Wolf has given the reader a liberal amount of wholesome mental pabulum. Many previously confused and confusing statements that have appeared in recent years, in the literature are carefully filtered out and clarified by this author. The exhaustive and carefully checked bibliography of 1,189 references is perhaps, the most completely and most thoroughly prepared list of references that has been published in recent years. Dr. Harold C. Wiggers considers this "the most extensive bibliography thus far published in this field". This unusual bibliography alone is worth the price of the book.

In addition, the reader may profit much, by reading and studying "Water Balance and Fluid Transfer", "Renal Physiology", "Dehydration and Hydration", and "Diuresis and Diuretics". "The Endocrines in Urinary Function" and "Antidiuresis and Antidiuretics" are interestingly and understandably presented. For all students and practitioners, and investigators interested in the study of urinary function of the kidney, this book is highly recommended as a useful and helpful work and they should not fail to read it once or twice.

ANNUAL REVIEW OF MEDICINE: Volume I. W. C. Cutting, M.D., Editor, H. W. Newman, M.D., Associate Editor. 484 pages. Annual Reviews, Inc., Stanford, California, 1950. Price $6.00.

As stated in the preface, the purpose of the *Annual Review of Medicine* is to incorporate in one volume the various fields of medicine under the editorship of well-known men. It is hardly necessary to mention all of them; however, among the contributors are Drs. Eusterman, Balfour, DeBakey, and J. A. Paulson, who need no further introduction.

The physician will find his money and time well spent in acquiring a copy of this Review for his library.

THE MERCK MANUAL OF DIAGNOSIS AND THERAPY, A SOURCE OF READY REFERENCE FOR THE PHYSICIAN: Eighth edition. 1592 pages. Merck and Co., Inc., Rahway, N. J., 1950. Price, Thumb-Index, $5.00; regular edition, $4.50.

This compact volume of 1,592 pages supplies the physician and medical student with easily read and understood medical information, with emphasis on diagnosis and treatment, including preventive medicine, immunization measures, as well as laboratory tests, prescriptions, medical emergencies, poisoning and venomous bites, electric shock, and drowning.

All the more common diseases are satisfactorily and clearly discussed. This edition is a great improvement over all previous editions.

This very complete manual is highly recommended to all medical students and practicing physicians, internes and residents. The price of this book is reasonable and it will have a wide sale among the many medical students and nurses, as well as among general practitioners of medicine.

PATHOLOGY LN GENERAL SURGERY: Paul W. Schafer, M.D., Professor of Surgery, University of Kansas School of Medicine. 581 pages, profusely illustrated in color and in black and white. The University of Chicago Press, Chicago, 1950. Price $17.50.

The reviewer has read and reread the work and admired the illustrations and their descriptions. To begin to describe this beautifully illustrated volume will take more space than is available.

Some of the examples found in the text are the carcinoma of the lip, opposite page 241, and, below it, the microphotograph of the biopsy material. On page 242 there is a large ulcerating carcinoma of the tongue, with an x-ray illustrating extensive bone destruction. Continuing on to page 571, the medical man, the surgeon, and the pathologist will find descriptions and illustrations, in black and white and in color, covering the entire alimentary tract. The organs of the rest of the body are similarly illustrated and described.

The reviewer is enthusiastic and would like to see many more such volumes printed. The work is highly recommended.

THE ANTHISTAMINES, THEIR CLINICAL APPLICATION: Samuel M. Feinberg, M.D., Saul Malkiel, Ph.D., M.D., Alan R. Feinberg, M.D., Northwestern University Medical School. 291 pages. The Year Book Publishers, Inc., Chicago, Ill., 1950. Price $4.00.

Because of the prevalence of so many complaints related to allergy and its effects, in a variety of manifestations, and since the pioneering work of Fourneau, Bovet, Staub, Halpern, and the continued interest of many American investigators in the field of "antihistaminics", such a small volume as "The Antihistamines" will be gladly received by the profession. The authors have given the readers much helpful information with concise clarity, and readily understandable discussions. The bibliography of 586 references should be helpful to those wishing to refer to the original articles. Forty-three pages (232-275) are taken up by the references and 14 pages by the index.

Included in the discussions are "Experimental Studies" and consideration of histamine, "Chemistry of Anthihistamines", "Pharmacology" and experimental hypersensitiveness, respiratory allergy dermatoses, administration and dosage of antihistamines, toxic effects, and an "appendix" of all available antihistaminic preparations with trade names, ingredients, and name of manufacturer.

This little work should be very helpful to all physicians interested in the use of antihistamines for the many allergic manifestations presented by their patients.

SECRETORY MECHANISM OF THE DIGESTIVE GLANDS: B. P. Babkin, M.D., D.Sc., LL.D., F.R.S.C., Late Professor of Research Physiology, McGill University, Montreal, Canada. Second edition, revised and enlarged, 1027 pages, 233 illustrations. Paul B. Hoeber, Inc., New York, 1950. Price $20.00.

The late Dr. Babkin brought the second edition of this important work on gastric physiology up to date, adding many new observations and experiments in line with advanced research. Wolf and Wolff's studies, duplicating those of Beaumont with newer concepts, are incorporated so that the physician or research worker will have a ready reference to the physiology of the secretory mechanism of the gastrointestinal tract in this volume.

A section on the action of drugs and the endocrine glands on gastric and intestinal secretions adds to the usefulness of the book.

The publishers are to be congratulated on the style, printing and illustrations. The work is highly recommended for the library of the medical student and the physician.

PRIMER OF ALLERGY: Warren T. Vaughan, M.S., M.D., Richmond, Va. Revised by J. Harvey Black, M.D., Dallas, Texas. 175 pages. C. V. Mosby Co., St. Louis, Mo. 1950. Price $3.50.

This small volume is a companion piece to the larger volume by the same author. This brief epitome on allergy "Man's White Elephant", in plain language, with cartoons illustrating some of the interesting problems, gives considerable helpful information for the sufferers, the patients and their lay friends. It makes easy reading for the young and old.

"Not all cases of urticaria, hives, nettlerash are of allergic origin". Focal infection, and purely emotional factors account for a certain proportion. Furthermore "these other factors may play a part in cases primarily associated with allergy", states the author. The author further adds that "allergists prefer to speak of relief" and have some hesitancy in using the word "cure"!

Chapter 11 includes "general orders, directions to the patient" which should be helpful. This little book may be safely recommended to all patients suffering from allergic conditions.

AN ATLAS OF HUMAN ANATOMY. Barry J. Anson, Ph.D., Professor of Anatomy, Northwestern University Medical School. 518 pages, profusely illustrated in color and black and white. W. B. Saunders Co., Philadelphia, 1950. Price $11.50.

Here is a book on anatomy which is one of the most useful of the texts on this subject. Students of medicine collaborated with the artists in the preparation of the illustrations.

Students preparing for examinations in anatomy or at the dissecting table will find much of value in this volume to guide them to a better understanding of the human body.

The surgical specialists, as well as the general surgeon, may benefit greatly by glancing through the beautiful and perfect illustrations by well-known artists.

The text is clear and the paper of superior quality — factors which make easy reading of the book.

Both the author and the publisher have done a splendid job in bringing out this volume.

CURRENT THERAPY 1950: Latest approved methods of Treatment for the Practicing Physician: Editor, Howard F. Conn, M.D., and 12 consulting editors. 736 pages. W. B. Saunders Co., Philadelphia. 1950. Price $10.00.

The contents of this excellent 1950 review of current therapy include many helpful and informative contributions by experts in their respective fields of special interest. Of particular interest to gastroenterologists and clinicians are the brief, but adequate discussion on "Amebiasis" by Spellberg; dysentry food poisoning, regional ileitis, chronic ulcerative colitis, by Joseph B. Kirsner; appendicitis by Cole, Keith, Keith S. Grimson; cholelithiasis, cholecystitis and pancreatitis by J. Edward Berk; tumors of the stomach by George T. Pack; hepatitis and cirrhosis of the liver by Richard B. Capps; tumors of the intestines by D. E. Clark; peptic ulcer by Julian M. Ruffin; fissure-in-ano, fistula-in-ano and hemorrhoids by C. E. Pope; gastritis by E. B. Benedict; megacolon and colitis by J. A. Bargen; constipation and diarrhea by P. W. Brown and J. L. Horner.

Many other interesting sections on therapy in "abridged abstract form" are included in this compact volume of 736 pages such as: diabetes mellitus by E. P. Joslin, Duncan, Root, and Wilder; hyperinsulinism by J. W. Conn; sprue, pellagra, scurvy, beriberi, and nutritive deficiency by James S. McLester; sickle cell anemia and agranulocytosis by Roy R. Kracke; hemolytic anemia by William Dameshek; Hodgkin's disease, lymphosarcoma, multiple myelomae and leukemia by M. M. Wintrobe; Splenomegaly and hemophilia by S. P. Lucia; coronary thrombosis by Harry Feil, William D. Stroud, and E. S. Michol; intestinal parasites by Meleney, Faust, and H. Most; tularemia by Foshay, etc. This volume is recommended for students, interns, residents and general practitioners of medicine.

PERSONALITY IN PEPTIC ULCER: A. J. Sullivan, M.D., Head of Section on Gastroenterology, Ochsner Clinic, and Thomas E. McKell, M.D., Member of Section on Gastroenterology, Ochsner Clinic. 112 pages, 13 illustrations. Charles C. Thomas, Springfield, Ill., 1950. Price $3.00.

In this small but comprehensive book the authors stress the psychosomatic and neurogenic factors in ulcer of the stomach and duodenum. It is highly recommended by this

reviewer for supplementary reading to those physicians who still cling to the old theory of ulcer formation.

The illustrations are ingenious, the type is clear, and the entire book well prepared.

MEDICAL MANAGEMENT OF GASTROINTESTINAL DISEASES: Garnett Cheney, M.D., Clinical Professor of Medicine, Stanford University, Medical School. 478 pages. The Yearbook Publishers, Inc., Chicago, Ill. Price $6.75.

This is an excellent practical small compact volume, giving a very informative and instructive review of many of the more common gastrointestinal disorders and their medical management. Among the interesting subjects covered are: Examination of the patient, clinical laboratory procedures, endoscopy and biopsy examinations, sore mouth and tongue, loss of appetite, and excessive hunger, indigestion and belching and heartburn, hematemesis, peptic ulcer management, upper abdominal discomfort, abdominal pain and colic, management of liver disease, abdominal distress and distention, abdominal tumors, constipation, chronic diarrhea, colitis, and food allergy.

This small epitome of gastrointestinal disorders and medical management should be of interest and of some aid to medical students, internes, nurses, and general practitioners, for quick reading.

GASTRITIS: E. Vidal-Colomer, M.D., Professor adjunto de la Escuela Medico-Quirurgica de Patologia Digestiva del Hospital de la Santa Cruz y San Pablo, de Barcelona, Jefe del Departamento de Enfermedades del Aparato Digestivo de la Clinica de Patologia General de la Universidad de Barcelona. 194 pages. Editorial Pas Montalvo Madrid, 1951

It is unfortunate that this book is only in Spanish because this subject has been pushed around in this country for years without getting anywhere in substantial ways. In Europe the subject of gastritis has had more extensive study, both clinically and pathologically, than here. Several decades back the diagnosis of gastritis was commonly made here. As time went on, especially because of better diagnostic work and the unfoldings of surgery, the diagnosis became unpopular. That is the way it went for years until the gastroscope came along. This awakened interest again and a controversy developed based on pathological appearance and a division of so-called chronic gastritis into the three forms of simple, hypertrophic and atrophic. This controversy is still raging on the basis that the pathology cannot be accurately judged by gastroscopic appearance even when assisted by test meal examination, that appearance changes from hour to hour, and some even claim that the term gastritis should not be employed.

If one reads this book one has to agree that "Gastritis" is an entity. Even if it is intimately bound up with referred, reflex, nervous states of the body which act on the stomach as an organ; the pathological and gastroscopic parts of the subject are exceptionally well done. The rugal changes as noted by x-ray showing that segmental lesions are common is quite an improvement upon any writings the reviewer is aware of in the English language; and the same may be said of the superficial ulcerated lesions that chipping-off phenomena (erosions) that has given positive occult blood findings that have been confused with true peptic ulcers.

Professor Vidal-Colomer has written a perfect exposition of the subject and he is to be congratulated. As the reviewer started out with, it is unfortunate a copy in English is not available for this country.

ANGINA PECTORIS AND MYOCARDIAL INFARCTION, with special reference to the autonomic nervous system. Heyman R. Miller, M.D., Attending physician, Sydenham Hospital, Assistant Professor of Medicine, New York Postgraduate Medical School and Hospital. 336 pages. Grune and Stratton, New York, 1950. Price $8.75.

The author of this work had previously published several other interesting books including publications in 1939 and 1942. In this present volume he expertly discusses the general clinical aspects, and the physiological aspects of angina pectoris and myocardial infarction Informatively presented are the autonomic regulation of circulation; autonomic pathways for cardiac pain; reference, distribution and simulation of anginal pain; concept and mechanism of pain; "Psychosomatic disorders of the heart"; and "Treatment of angina pectoris and myocardial infarction". "Sugical Treatment" is concisely and adequately presented. Very helpful bibliographic references follow the various chapters. The book is easily read, and is highly recommended to all physicians, surgeons, internes, and students who should carefully digest its contents and follow its teaching! This is a small but practi-

cal review of an all important subject. Angina pectoris and myocardial infarction, coronary occlusion and thrombosis, a killer of men, all too often most unexpectedly and tragically sudden, still presents many problems that are confusing to all physicians since Archangelus Piccolomini in 1586 gave it as his opinion that "obstruction or obliteration of the little vessel or nerve to the heart, occurring suddenly, is the cause of sudden unexpected departure from this world"!

TEXTBOOK OF ROENTGENOLOGICAL DIFFERENTIAL DIAGNOSIS. (LEHRBUCH DER ROENTGENOLOGISCHEN DIFFERENTIAL DIAGNOSTIK). Werner Teschendorf. Vol 1: Diseases of the chest. 780 pages, 865 illustrations. Second edition. Georg Thieme, Stuttgart. Grune & Stratton, New York. Price $20.20.

This is the first volume of an elaborate textbook on roentgenological diagnosis. Teschendorf has used a new approach in dealing with his material by taking the leading roentgenological signs to discuss the findings and their evaluation, for the final diagnosis. In the section on chest pathology we find chapters on dense shadows of one part of the chest, such as shadows of the pleura, of parts of the lungs, of interlobar spaces, sharply defined and unsharply defined shadows. The differential diagnosis of small patches and larger ones, and the cavities in the lungs are special chapters. One part is devoted to the hilar region, one to the mediastinum.

Chapters are devoted to the size, shape and position of definite parts of the heart. The kymogram and its value for the diagnosis is discussed. Other chapters deal with electrocardiography and its relationship to roentgenology. Of interest are the chapters on the demonstration of calcified coronary arteries and the calcification of the valves of the heart. For the roentgenological heart function the following tests are discussed: 1. the determination of the heart volume, 2. the kymogram, 3. tests by Abreu, 4. tests by Buerger.

The last two parts concern the examination of the esophagus and the diaphragm. The pathological condition of both organs is discussed.

The entire book has 865 illustrations, all of them excellent reproductions, more illustrations than pages. This is a great asset, especially for those readers who are not masters of the German language. All modern methods, such as kymography and tomography, are widely used and their importance in the diagnosis is stressed.

The references are at the bottom of each page and cover the entire world. Especially well represented is the work done in the USA. The print is excellent and the publishers can be highly praised for the print and paper used in their work. Considering that a great deal of the original material has been lost, we want to congratulate Dr. Teschendorf for presenting his subject so well. This book can be highly recommended to all those who are interested in internal medicine and its roentgenological problems.

PENICILLIN—ITS PRACTICAL APPLICATION: Edited by Professor Sir Alexander Fleming, M.B., B.S., F.R.C.P., F.R.C.S., F.R.S., Professor Emeritus of Bacteriology, University of London. Second Edition. 491 pages. Butterworth & Co., Ltd., London; The C. V. Mosby Co., St. Louis, Mo., 1950. Price $7.00.

In this second edition the contributors have revised and rewritten their section, and some new chapters have been added bringing this edition up to date, and more informative and instructive than the first edition of three years ago. Only brief discussions, in Appendix B (pages 466-471), appear in this edition, of chloromycetin (chloramphenicol) and aureomycin (duomycin). The third edition will no doubt include more detailed discussions of aureomycin, chloromycetin, and the other newer antibiotics such as terramycin and neomycin.

This volume, of course, is concerned chiefly with penicillin, its manufacture, pharmacology, and its practical uses in the various infectious diseases, plastic surgery, eye infections, dermatologic conditions, diseases of cattle, dogs, cats, and birds. There is a chapter on dental and oral infections. Streptomycin is briefly, but adequately and satisfactorily presented by Crofton and Daniels (pages 400-424).

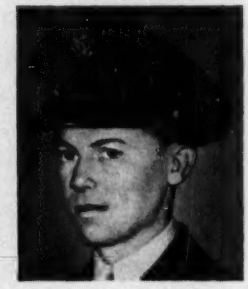
This book is an excellent resume and epitome of present day knowledge of penicillin and some of the newer antibiotics.

Medal of Honor

Major General William F. Dean, Berkeley, Calif.—Medal of Honor

Sergeant Charles Turner, Boston, Mass.—Medal of Honor

Lieutenant Frederick Henry, Clinton, Okla.—Medal of Honor



Private First Class Melvin Brown, Mahaffey, Pa.—Medal of Honor

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But this year remember another star, too—the one on the Medal of Honor. And make a place in your heart for the brave, good men who've won it. Men who, oftener than not, made the final, greatest sacrifice—so that the stars on your Christmas tree, and the stars in your country's flag, might forever shine undimmed.

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*Peptic Ulcer. A. C. Ivy, M. I. Grossman and W. H. Bachrach, Blakiston Publishing Co., Phila., 1950.

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*S. Krasnow, F. Sorkness and L. L. Hardt, Comparison of Effectiveness of Various Antacids on Gastric Acidity. Am. J. Dig. Dis. [illegible] (1950).

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*[illegible] J. Raimondi, Treatment of Duodenal Ulcers with Desiccated, Defatted Duodenal Powder. Permanente Foundation Med. Bull., 8:4 (October), 1950.

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*Medical Management of Gastrointestinal Disorders. Garnett Cheney, Yearbook Publishers, 1950.

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*M. H. Streicher, J. Lab. Clin. Med. 33, 1633 (1948).

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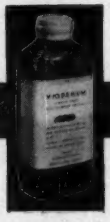

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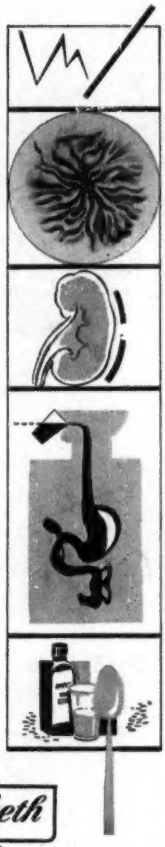

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